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MEDICAL DISEASES

OF

INFANCY AND CHILDHOOD

BY

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SECOND EDITION REVISED WITH ADDITIONS

BY

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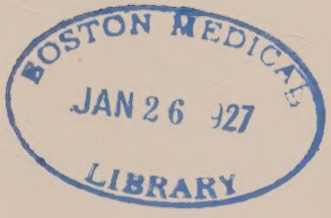

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PREFACE TO THE AMERICAN EDITION.

It has been the object of the reviser, in preparing this book of Dr. Williams' for its second American issue, to adapt it more closely to the special requirements of this country.

The original text is here presented entire, no part having been omitted. The additions, requisite to bring the work up to date and to represent American views, have been enclosed in brackets. The prescriptions have been scattered through the text for convenience of connected reading. They have been conformed to the United States Pharmacopeia. The reviser however would not imply that a specific prescription refers to a specific instance. To avoid the possibility of misinterpretation the Appendix of Prescriptions as a whole is retained as in the original edition.

Particular attention has been given to infant-feeding, both on account of its importance and because in this respect the original edition seems to depart most from American ideas and standards.

The reviser wishes to acknowledge his indebtedness to the various authors consulted, especially to Dr. T. M. Rotch. He wishes particularly to express to Dr. Williams the interest and profit found in the study of his work, and trusts that the additions made may be of value to the public.

F. S. C.

Chicago, Ill., August, 1900.

PREFACE TO THE ENGLISH EDITION.

THE object of this handbook is to give to young practitioners of medicine, and to those who have not previously paid much attention to the subject, a guide to the clinical study of disease as it occurs in infancy and childhood.

No attempt has been made—nor was it, indeed, possible within the space at my disposal—to attain an ideal completeness in the enumeration of all the forms of disease which may occasionally be met with in infants and children. Pathological processes are essentially the same in children as in adults. The differences to be observed are traceable in the main to two causes. In the first place, the organism in childhood is growing, and while it is peculiarly vulnerable to external agencies, it possesses also a special power of adaptation and recuperation. In the second place, the organism in childhood has not yet acquired immunity to the acute specific infectious diseases which are, as a matter of fact, responsible for a very large part of the enormous mortality of the early years of life.

I have not deemed it to be my duty to attempt to describe fully diseases which present symptoms similar at all ages. My object has been rather to indicate the special incidence of disease in childhood; to elucidate as far as possible the causes of this special incidence; to point out the peculiarities which the circumstances of child-life impress upon familiar diseases; and to detail the treatment rendered appropriate by the nature of the disease itself and by the peculiar susceptibility of the growing organism. While it would be difficult to mention any disease—except, perhaps, rickets—which is peculiar to childhood, yet certain morbid processes present special features or a peculiar distribution in childhood, and others, common at that period, are rare in adult age. Moreover, the relative importance of diseases varies greatly at different ages. Diarrhœa, for instance, which at adult ages and in temperate climates is usually a trivial,

seldom a fatal, affection, is in infancy and early childhood the most deadly of all disorders.

In discussing the subject of treatment, prominence has been given to the rational basis afforded by pathology, and by clinical observation. While it is hoped that few remedies which experience has proved to be valuable have been omitted, it has not been thought worth while to compile long lists of drugs which have been recommended and employed without a reasonable measure of success. I have been guided throughout, both in clinical description and in the treatment recommended, by experience gained during many years' service at the East London Hospital for Children, Shadwell. The opportunities for observation there afforded are very extensive, but they have failed me in regard to two forms of disease. Malaria is seldom seen in London in children, but I have been so fortunate as to obtain the assistance of Dr. Manson, who has been good enough to revise the chapter on Malarial Fever. Hydatid disease also is extremely rare in this country, and I am greatly indebted to my friend, Mr. G. Twynam, formerly Surgeon to the Prince Alfred Hospital, Sydney, New South Wales, who has read the chapter on this subject, and has made many valuable suggestions which are embodied in the text. In conclusion, I must express my acknowledgments to Miss Mary Gordon, L.R.C.P. & S. Ed., who has read the proofs, and assisted me in seeing the volume through the press.

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MEDICAL DISEASES

OF

INFANCY AND CHILDHOOD.

CHAPTER I.

INTRODUCTORY.

The Ages of Childhood—Growth—The Mortality of Childhood—Its Causes—Influence of Sex—Sleep—Clothing—Baths—Change of Air.

The Ages of Childhood.—Within the period of childhood are included two of the ages of man—infancy and childhood proper.

Infancy is used often in a loose sense to signify the period of early childhood. Etymologically the word "infant" signifies a child which has not yet acquired the art of speech.[1] It was extended by the Romans themselves to include children up to the age of seven years. For medical purposes it is better to define infancy as the period during which the child suckles, since this is a well-defined epoch of life which comes to a natural termination with the establishment of the first dentition. It corresponds with the *Säuglingsalters*, or suckling age, of German writers, and may be held to extend in the healthy child to the end of the first year, at which age, or soon after, the first molars are cut. It is convenient to recognize as a separate age or period of life the first two or three weeks, during which the organism of the new-born infant undergoes important adaptations to the new conditions of independent existence.

Childhood proper extends from infancy to puberty. It embraces the whole period of the functional activity of the first set of teeth, and the establishment, though not the completion, of the second dentition. In Rome the boy assumed the *togo virilis* at the age of fourteen or fifteen. In more northern countries puberty develops a little later, but in both sexes there are very considerable individual variations. Menstruation may begin as early as the tenth year—in

[1] *In*, not ; *fans*, present participle of *fari*, to speak. (Skeat.)

rare cases even earlier—or may be postponed to the seventeenth, or even later.

Growth.—During the first few days after birth there is a slight decrease in weight, which is not regained until about the end of the second week. After this, in a healthy infant, the increase in weight is steady; but the rate of increase declines progressively. At the end of the fifth month the weight will be about twice that at birth, but it will not reach thrice the weight at birth until the child is a year old. After weaning, if the child is wisely fed, and remains in good health, the weight at the end of the second year will be about four times that at birth. If we take the average weight of an infant at birth to be 7½ lbs. (3,000 grammes; male 3,200, female 2,900)[1] we shall, by calculation, obtain the following weights:—At five months 15 lbs. (6,000 grammes); at one year 22½ lbs. (9,000 grammes); figures which agree very closely with the averages obtained by Sutils[2] from actual weighings:—At birth 3,000 grammes; at five months 6,250 grammes; at twelve months 9,000 grammes; at twenty-four months 11,550 grammes.

[The average weight at one year, given above, is higher than that observed by most American authors, who place it at about 20 lbs. This average, however, is based on statistics taken from hospital children; observations made in private practice will range somewhat higher on account of the better care and feeding of the latter class of children. The value of weekly observations on the weight of infants during the first year cannot be overestimated. The healthy infant should gain, at least, four ounces weekly during the first six months; in the second six months it may be slightly less. If the infant be weighed more than once a week, we must be prepared to see marked variations in the rate of gain, *e. g.*, a gain of .5–1 ounce the first 2–3 days of the week, and of 3–5 ounces the last 3–4 days, and *vice versa*.

Except in special cases, therefore, it is wiser to weigh but once a week, especially if we have to do with an infant at the breast of a nervous mother, whose milk may be upset by too critical an analysis of the weight-curve.

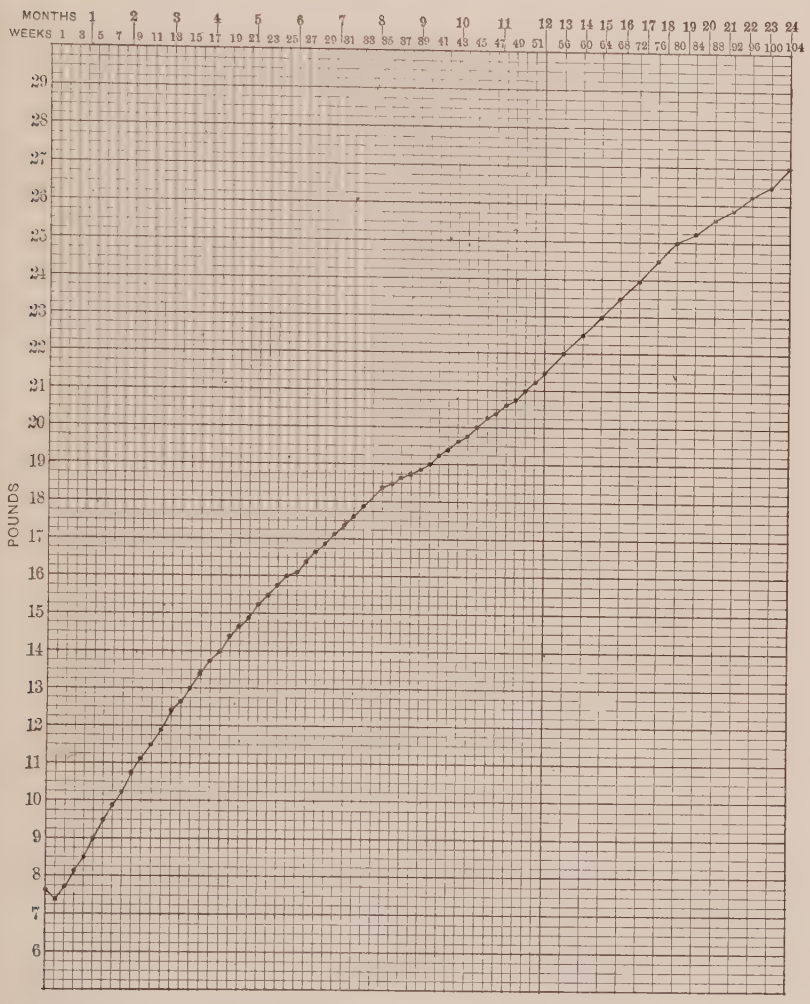
The following chart (Fig. 1), by Griffith, is of practical convenience in recording the infant's weight.]

The increase in height is also regular, and follows a similar law—that is, the rate is rapid at first, and gradually declines. This has been expressed conveniently by Liharzik, who found that on the average an increase in height of 7½ centimetres (3⅓ inches) took place in periods progressively longer as the infant became older; thus this increase took place in succeeding periods of one month, two

[1] Ballantyne, "Introd. to Diseases of Inf.," Edinburgh, 1891, p. 194.
[2] Sutils, "Guide Prat. des Pesages," Paris, 1889, p. 58.

months, three months, four months, five months, six months, and so on. The following table, extracted from one compiled by Mr. Charles Roberts,[1] shows the average height and weight of children

FIG. 1.



of both sexes from five years old upwards. Under that age the rate of growth is the same, though boys are a little taller and heavier than girls. From five to ten years boys grow rather more rapidly than girls, while from ten to fifteen the reverse is the case, owing to

[1] "Med. Inspect. and Phys. Education in Schools," London, 1895.

a diminished rate of growth in boys. After fifteen years of age girls grow very slowly, and their full stature is gained at twenty years, three years earlier than in males.

HEIGHT AND WEIGHT OF CHILDREN FROM 5 TO 15 YEARS OF AGE.

Age last birthday.	Height without shoes in inches.		Weight with clothes in lbs.		Ratio: Weight divided by height.	
	M.	F.	M.	F.	M.	F.
5–6	41.0	40.8	39.9	39.6	.97	.97
6	44.0	42.6	44.4	42.4	1.01	1.00
7	46.0	44.5	49.7	46.7	1.08	1.05
8	47.1	46.6	54.9	52.2	1.16	1.12
9	49.7	48.7	60.4	55.5	1.22	1.14
10	51.8	51.1	67.5	62.0	1.30	1.21
11	53.5	53.1	72.0	68.1	1.35	1.28
12	55.0	55.7	76.7	76.4	1.39	1.37
13	56.9	57.8	82.6	87.0	1.45	1.51
14	59.3	59.8	92.0	96.7	1.55	1.62
15	62.2	60.9	102.7	104.8	1.65	1.72

[It is interesting to note in this connection tables of weight and height in American children compiled by Rotch and Holt from the observations of Bowditch.

Age.	Sex.	Height in inches.			Sex.	Weight in pounds.[2]		
		Williams.[1]	Rotch.	Holt.		Williams.	Rotch.	Holt.
5-6 years.	M.	41.0			M.	39.9		
	F.	40.8			F.	39.6		
6 "	M.	44.0	43.75	44.1	M.	44.4	45.07	45.1
	F.	42.6	43.35	43.6	F.	42.4	43.18	43.8
7 "	M.	46.0	45.74	46.2	M.	49.7	48.97	49.5
	F.	44.5	45.52	45.9	F.	46.7	47.30	48.0
8 "	M.	47.1	47.76	48.2	M.	54.9	53.81	54.5
	F.	46.6	47.58	48.0	F.	52.2	51.56	52.9
9 "	M.	49.7	46.69	50.1	M.	60.4	59.00	60.0
	F.	48.7	49.37	49.6	F.	55.5	57.00	57.5
10 "	M.	51.8	51.68	52.2	M.	67.5	65.16	66.6
	F.	51.1	51.34	51.8	F.	62.0	62.23	64.1
11 "	M.	53.5	53.33	54.0	M.	72.0	70.04	72.4
	F.	53.1	53.42	53.8	F.	68.1	68.70	70.3
12 "	M.	55.0	55.11	55.8	M.	76.7	76.75	79.8
	F.	55.7	55.88	57.1	F.	76.4	78.16	81.4
13 "	M.	56.9	57.21	58.2	M.	82.6	84.67	88.3
	F.	57.8	58.16	58.7	F.	87.0	88.46	91.2
14 "	M.	59.3	59.88	61.0	M.	92.0	94.49	99.3
	F.	59.8	59.94	60.3	F.	96.7	98.23	100.3
15 "	M.	62.2		63.0	M.	102.7		110.8
	F.	60.9		61.4	F.	104.8		108.4

It would appear that American children are on the whole both taller and heavier than their English cousins. Observations by Porter, however, on St. Louis children show them to be lighter in weight than those given above.]

[1] Without shoes. [2] With clothes.

The Mortality of Childhood.—It is not uncommon to find the diseases of childhood made light of, and to hear the "therapeutics of the nursery" spoken of with a certain scorn. The inhabitants of the nursery, however, are numerous. About one-third of the population is under fifteen years of age; a quarter under ten, and these numerous inhabitants of the nursery are the hope of the future.

The mortality among infants and children is enormous. More than two-fifths of the deaths in England and Wales during the ten years 1881–1890, occurred among children under the age of ten years. The greater part of this huge mortality took place under five years of age. With a mean population under five years of three and a half millions, in round numbers, the deaths numbered two millions. In the same decennium there were nearly nine million births and over two million deaths. The annual death-rate per 1,000 at all ages was 19.08; under five years it was 56.82. We must pass to the age period sixty-five to seventy-five—the Psalmist's span of life—before we again find the death-rate rising to so high a level. As the number of deaths in the first five years of life is greater than at any other period of life of the same duration, so the number of deaths in the first year of life is greater than in any of the subsequent years. Nearly a fourth of all the deaths registered in the decennium were those of infants under one year—a million and a quarter out of a total of five millions and a quarter. In the German Empire in the two years 1892–93 the deaths in infants one year old and under exceeded a third of the total number of deaths. It will be instructive to indicate briefly the main causes to which this excessive mortality in infants and children was attributed.

If the annual mortality per 1,000,000 living at all ages and at childish ages in England and Wales be compared, it is seen that the rate in childhood in the decennium 1881–90 was in excess in the following classes of causes :—

	0–5.	5–10.	10–15.	All ages.
Respiratory diseases	12,966	853	213	3,729
Acute specific diseases	9,130	1,839	541	1,667
Nervous diseases	8,337	578	320	2,592
Diarrhœal and digestive diseases	7,308	279	177	1,778
Tubercle	4,499	844	827	2,420
Violence	1,142	325	263	648

The influence of infectious processes is even greater than appears from the table, for under the head of nervous diseases are included cases of intracranial tubercle, and diarrhœa accounts for considerably more than half the deaths due to diarrhœal and digestive diseases.

With regard to diseases of the respiratory system also there can be little doubt that a very large proportion was due to infective forms of broncho-pneumonia. Further it will be seen that the enormous excess in the death-rate in childhood is due almost entirely to its excess in children under five years. In the other two quinquenniads of childhood the death-rate is below the average, with the single exception of the rate from acute specific diseases at the age-period five to ten. The classes of disease the death-rate from which in childhood is lower than the average at all ages are, in fact, few. The most important are :—

	0–5.	5–10.	10–15.	All ages.
Urinary system	193	92	62	435
Circulatory system	134	149	258	1,576
Cancer	20	10	11	589

Sex.—Since the generative system is very far from its full development in early childhood, it might have been expected that the death-rates of the two sexes would have shown little or no difference. This, however, is not the case. The common opinion of matrons that male are more difficult than female infants to rear is borne out by statistics. The annual mortality among males is greater than among females at all age-periods except from ten to twenty. The disparity is greater under five years of age than at any other period, except at sixty-five and upwards : males, 61.69 per 1,000 ; females, 51.99 per 1,000. The death-rate under one year of age is, per 1,000 births, males 155, females 128. The mortality in the two sexes per 1,000,000 living from the causes in respect of which childish mortality is in excess of the average, is shown in the following table :—

	0–5.		5–10.		10–15.		All ages.	
	M.	F.	M.	F.	M.	F.	M.	F.
Respiratory diseases	14,141	11,799	846	821	211	215	4,096	3,382
Acute specific "	9,008	9,252	1,735	1,939	495	583	1,694	1,641
Nervous "	9,451	7,231	601	556	322	318	2,804	2,392
Diarrhœal and digestive diseases	8,007	6,614	263	293	188	168	1,875	1,687
Tubercle	5,010	3,991	815	872	627	1,026	2,622	2,229
Violence	1,266	1,019	451	199	435	91	968	347

These statistics and others quoted above are compiled from the Decennial Summary, 1881–90, of the Registrar-General.

It will be seen that the number of deaths attributed to *acute specific diseases* is greater among girls than among boys throughout childhood ; that at ages five to ten years diarrhœal and digestive diseases are rather more fatal to girls ; and that tubercle, which is rather

more fatal to them than to boys at the age-period five to ten, is much more fatal to them at the age-period ten to fifteen. With these exceptions, however, the death-rate of boys is higher at each age from each of the most important classes of diseases. Under five years the greater mortality of boys from respiratory, nervous, diarrhœal, and tuberculous diseases is very remarkable. No adequate explanation of this disparity, which appears in all the statistics with which I am acquainted, has been given. It has been urged that the fact that male infants are on the average heavier than female renders them more liable to injury at birth, but this can hardly be held to account for their higher mortality from respiratory and diarrhœal diseases, and is no explanation of the fact that the death-rate of boys from tubercle is greater by one-fourth. Nor does the suggestion that males are more exposed to the vicissitudes of life appear a sufficient explanation, since there is very little difference in the management of children of either sex under five years of age; and at the age-period ten to twenty, when this cause might be imagined to be most operative, the difference in the death-rate is in favor of males.

Sleep.—An infant in good health sleeps seven, eight, or nine hours by night, and for an hour or more between each suckling by day. The cradle in which it passes so much of its life during the first year is, therefore, deserving of attention. It should be light and easily cleaned. Many cradles are much too deep. This leads the nurse to wrap the infant in a blanket, and then put a blanket and coverlet over the sides of the cradle; as there is usually a curtain or hood at the head, the infant lies sweating at the bottom of a cavity closed below and very much obstructed above. All bed-clothing should be of woollen, and if the outer covering must be ornamental a colored or embroidered blanket should be selected. An infant should not sleep in the same bed with its mother or nurse. Apart from the fact that a large number of infants are annually suffocated or crushed by being overlaid in bed, the practice is objectionable because the infant is apt to be overheated and deprived of its share of air by being buried under the bed-clothes. It has the further drawback of tending to encourage the vicious practice of permitting the infant to suckle at odd times, or at short intervals, during the night.

During the second and third years of life a healthy child will continue to take part of its sleep by day, and the custom of making a child at least lie down, if it do not sleep, in the middle of the day, until five or six years of age, is a good one. The night's sleep at three years of age should be for about eleven or twelve hours. If left to itself the child will as it grows older, begin to shorten its night's sleep by waking up a little earlier in the morning. It is much better to allow this shortening of the hours of sleep to be thus spontaneously effected than to permit the child to sit up later in the

evening. Most children of school age, especially among the poorer classes, do not get enough sleep. A child of ten years needs at least ten hours, which means fully eleven hours in its bedroom; if breakfast is to be at eight a.m. the child should, therefore, "go to bed" at nine p.m. Every child should have a separate bed, and, after the age of ten years, a separate bedroom or cubicle.

The nursery in which an infant lives so large a part of its time should be spacious, and well warmed and ventilated. It should be furnished simply, all floor coverings should be easily removed, and the whole room kept scrupulously clean. The floor should receive special attention, for the infant when it begins to crawl will carefully examine every object which it comes across by putting it into its mouth.

After the first fortnight a healthy infant should spend at least three hours a day in the open air, and, short of falling rain or snow, there are in temperate climates no weather conditions which should keep it indoors.

Clothing.—It seems to be the rule in tropical climates to dispense the indigenous infants and young children from clothing of any kind; but it is a curious circumstance that in temperate and cold regions the practice appears to have been universal of wrapping infants in swaddling clothes which seriously impeded the movements of the lower limbs, and, under some systems, of the upper also. This custom probably had its origin in the recognition of the fact that one of the primary needs of infancy is warmth. Provided that it be kept warm and supplied with its natural nourishment an infant will commonly flourish under circumstances in other respects most adverse, showing an imperviousness to injurious influences and an immunity from many infectious disorders, which are really remarkable.

The clothing of an infant should be, in the main, of woollen materials,[1] but the custom of using a garment of fine linen (cambric) or cotton next the skin of the trunk has much to recommend it. The use of a binder, either of woollen or knitted material, applied so as to cover the whole of the abdomen, is customary, but is more necessary in infants and children whose skirts have been shortened than in the young infant, which is usually enveloped in voluminous petticoats. The napkin to catch the urinary and alvine secretions should be of soft cotton diapering or towelling. It should be renewed as often as it is soiled, and all forms of waterproof retainers should be avoided, except under such special circumstances as a journey. When a child reaches the age of eighteen months or two years, the fault most often committed is to make its clothing too heavy, and too dependent by straps from the shoulders, while the belly and thighs are

[1] The belief that it is difficult to wash woollen materials without causing them to shrink is a myth fostered by incompetent laundresses.

often left uncovered or insufficiently protected. A binder, or soft woollen garment fitting closely to the belly, is an essential precaution at almost all seasons of the year in temperate climates. The legs should be covered by long knitted drawers, open back and front; and the arms by sleeves attached to the dress, or by a sleeved jacket. Even when the child is older parents are very apt to neglect to provide adequate clothing for the lower part of the body, while at the same time they often overload the chest.

Baths.—Great pains should be taken to keep an infant clean, but it is possible to bathe and wash too much. Under the head of the treatment of the specific fevers by baths will be found some observations on the very considerable effect on the body temperature which even lukewarm baths may have. In feeble infants the exposure necessary to give a complete bath may produce a degree of depression which should be avoided. In such cases the child should be washed piecemeal on the nurse's lap before a good fire. A healthy infant should be washed every morning; it is first lathered with a soapy flannel on the nurse's knee, then held in a sitting posture in the bath or basin and sponged rapidly. Many infants are all the better if this washing be repeated in the evening. The buttocks and perineum should be cleansed with hot water and soap after each action of the bowels, the parts dried with a soft towel, and dusted with starch powder. A full warm bath at about 90° F. may be given daily to a healthy infant at the age of six months, and the infant may gradually be accustomed to a lower temperature by making use of cool affusion at the moment of its being taken out of the bath. At or about the age of five years a cold bath may be agreeable to the child in warm weather, but if it show a dislike for the cold bath it is better to continue the use of warm water followed by cold sponging. In any case, the whole process of soaping, bathing, drying, and dressing should be carried through as quickly as possible in a warm room. In England the morning cold tub has been raised to the dignity of a national custom, but in childhood as in old age it is more often injurious than beneficial.

Change of Air.—If an infant reside in the country, annual change of air is certainly not necessary, and is frequently undesirable. During the first two years the nursery in which it lives is a more important source of well-being than change to the seaside. For town-bred infants a change to the country during the hot weather, when the air of large cities is charged with dust, is certainly desirable. Children of two years old and upwards who can run alone are greatly benefited by residence, for some months annually at least, in the country, where greater freedom can be allowed and where, in consequence, they spend the greater part of the hours of daylight in the open air. Children of a "scrofulous" disposition, and those who are anæmic, marasmic, or

ill-grown, commonly derive great advantage from residence for some months in every year at the seaside. For such children the east coast of England offers special advantages. The north coast of France possesses a similar, but somewhat milder, climate. To obtain the full advantage of sea air in scrofulous diseases, enlarged tonsils, and other disorders of the lymphatic tissues, it is necessary that the patient should reside within fifty yards of high water mark, and that the interspace should be free from buildings and other obstructions, or, as an alternative, that the hours of daylight should be spent on the beach. Children with a rheumatic or gouty tendency generally do better inland on a dry, porous soil. For children of a highly nervous organization the seaside is usually too stimulating. For them, at least, sea bathing is not to be recommended, and no child should be forced to bathe in the open sea against its will. The custom of permitting children at the seaside to spend many hours a day paddling with bare feet and legs is dangerous, and has been responsible for many attacks of diarrhœa and various congestive disorders of the viscera.

CHAPTER II.

CLINICAL EXAMINATION.

Clinical Examination of Infants and Young Children—General Observations—Physical Examination of the Upper Air Passages—Of the Chest—Of the Circulatory System—Of the Abdomen—Of the Head—Retraction of the Head—The Temperature in Infancy and Early Childhood.

Clinical Examination of Infants and Children.—In the treatment of the diseases of infants and young children the physician must rely almost entirely upon his own observations of the symptoms and physical signs presented by the patient. Infants can give no information directly, and the statements of young children as to the seat of pain or discomfort are commonly very indefinite and untrustworthy.

Careful inquiries as to the past *history* of the child should be made, and the value to be attached to the statements made must be estimated after taking into consideration the intelligence and mental organization of the mother or nurse. An irritable, hysterical, or unprincipled woman may grossly, if sometimes unconsciously, exaggerate or minimize the degree and amount of ill-health from which the patient has suffered. The family history will often throw much light on the constitutional peculiarities and tendencies which the child may be expected to have inherited to a greater or less degree. Undoubtedly, the early demise of many other children of the same family aggravates the prognosis, whether the deaths have been due to constitutional defect or to ignorance and incompetence in the mother. It is desirable to make a note as to all the previous pregnancies of the mother; a syphilitic taint may thus be suggested which might otherwise escape detection. Again, many deaths from intestinal disorders may point to radical defects in the sanitary surroundings, or in the method in which the mother cares for and feeds her offspring.

Statements as to the infectious diseases from which a child has suffered previously must be received with a certain amount of caution. With regard to measles, in particular, mistakes are often made; in mild cases of fever, with a rash resembling measles, medical assistance may not have been obtained, although the mother

may subsequently feel justified in making a most positive statement that the child has suffered from measles.[1]

An *infant*, especially if the disease be of chronic character, should first be seen dressed fully, because faults in dress are fertile causes of illness. Much may be learnt from the general appearance and movements of the infant. A healthy infant, when awake, is, except perhaps immediately after suckling, in almost constant movement; the hands and upper and lower limbs are continually in motion. Its gaze moves slowly and uncertainly from one object to another; it grasps a finger firmly, and shows a strong disposition to carry any object placed in its hand to its mouth, and is provoked to smile even more readily than to cry.

Short, sudden cryings, especially if they come on soon after feeding, and are accompanied by drawing up of the thighs to the abdomen, usually indicate gastric disturbance and flatulent colic. The infant will usually take the breast readily, even greedily, and is, therefore, often supposed to be hungry. Persistent crying, attended by some blueness about the lips, and disinclination to take the breast, will often be found to be due to intestinal disturbance, and will be followed by diarrhœa, or relieved by an evacuation produced by castor oil. A flushed and perspiring face, with increased rapidity of breathing, will suggest bronchitis; rapid, regular breathing, with an anxious expression and pallor or irregular flushing of the face, pneumonia. It would be easy to enlarge on the information obtained from a study of the physiognomy of disease in infants and young children, but personal observation alone can afford any profitable knowledge. Suffice it to say that the assistance to be obtained from a careful examination of the manner, attitude, and general appearance is never to be despised.

While the infant is being undressed, information may be obtained as to the existence of general or local tenderness, and as to the mobility of the limbs. The chest and abdomen should be quite denuded of clothes, including the belly band, and the infant should then be wrapped in a blanket. The front should first be examined with the infant lying on the nurse's lap. It should then be lifted up so that its belly rests against the nurse's chest and its head looks over her shoulder; the back is thus brought well into view. Or the infant

[1] Certain phrases are so commonly used by nurses and mothers of the poorer classes in Great Britain with fairly well-defined meaning that it may be worth while to mention some of them. Thus "windy spasms" signifies abdominal pain with eructation, or expulsion of flatus from the anus; "inward convulsions" usually colic, with spasmodic or jerky movements of the arms and drawing up of the leg, sometimes slight eclampsic or epileptic attacks, sometimes respiratory spasm; "convulsion fits" signify marked eclampsia as a rule; "taken off his feet," that a child who has once walked has ceased to care to do so, a common symptom of early rickets. The phrases vary in different localities, and it is of some importance to become acquainted with them.

may be placed face downwards on the nurse's lap, or on a bed with a pillow under its chest and belly. The infant should next be replaced on the lap and the napkin removed for inspection and palpation of the buttocks and anal region. Lastly, the mouth and throat should be examined in a good light, a manœuvre which commonly leads to crying, and which should, therefore, be deferred until the end of the examination. The whole should be done speedily but gently, and without jerks—*festina lente*. Auscultation of the chest and palpation of the abdomen ought to be accomplished before the infant begins to cry. Sudden movements, or anything like rough handling, will precipitate the almost inevitable protest from the patient; but firm palpation, if gently applied, is very well borne until some area of tenderness is reached, and valuable information is thus gained. It is, as a rule, waste of time to seek to conciliate a young infant, or to divert its attention. This duty is better relegated to the nurse.

With *children*, however, the opposite is the case, and every effort should be made to establish friendly relations or, at least, to disarm active opposition. The first essential is to give the child time enough to come to his own conclusions as to the unaccustomed visitor, and the interval may be utilized in gathering the history of the illness from the mother or nurse; this plan has the additional advantage of diverting her attention from the child, who is thus left quite undisturbed to complete his examination of the physician, which, it must always be remembered, must be allowed to precede the examination of the patient by the physician. In children under the age of five or six years, the routine to be followed in making a physical examination should be very much the same as for infants, and in every case every part of the person should be examined by the eye and touch. It is best, therefore, if no objection is raised, to have the child stripped and rolled in a blanket, and as many children very much dislike having their clothes taken off, it is often wisest to see the patient in his cot, which should be brought into a good light and one side removed. In children over eight to ten years of age the physical examination may, as a rule, be carried out as in adults.

In treating an infant or child presenting symptoms of catarrh or obstruction of the **upper air passage**, it is essential to make a careful examination of the pharynx. The patient should be seated in a good light on a stool or on the nurse's lap; the nurse, standing or sitting behind the patient, should grasp one of the child's wrists in either hand, and its back and shoulders should rest against her chest or arm, so that its head falls backward. The child should then be induced to open its mouth and, care being taken that the tongue is within the line of the teeth, the depressor should be rapidly introduced and the tongue pushed gently but firmly downward. A com-

plete but brief view of the palate, tonsils, and pharynx will thus be obtained before the child begins to swallow or cry. Prolonged inspection always causes much distress, and generally fails in its object. If the first glance does not give the desired information, it is better to wait a short time and repeat the manœuvre. In making applications to the throat the same method should be followed.

Laryngoscopical examination is, in infants and young children, exceedingly difficult owing to their restlessness and the small size of the parts, which in cases calling for such examination are usually swollen and more sensitive owing to the presence of catarrh. A small mirror must be used, and, at most, a very fleeting view of the larynx can be hoped for.

Laryngeal obstruction when it produces dyspnœa causes *recession* during inspiration of the tissues in the suprasternal notch, in the intercostal spaces, and of the lower part of the front of the chest. In young children, owing to the elasticity of the costal cartilages, the recession is most marked in the last-named situation, each inspiration being accompanied by a depression of the epigastrium and the lower part of the sternum and adjacent costal cartilages. In rickety children the area over which the recession occurs may be considerably larger, the whole of the lower part of the chest in front being drawn in, and the upper part of the sternum thrust out. The recession in this and the other situations mentioned is due to the increase in the negative pressure within the thorax with each inspiration owing to the obstruction to the entrance of air, and the forcible action of the muscles of respiration. At the lower part of the chest this is reinforced by the pull of the diaphragm on the ensiform cartilage and the lower costal cartilage.

When called to treat a child supposed to be suffering from disease of the **respiratory organs**, a thorough examination should be made on the first occasion. For this purpose the patient should, if possible, be seen under natural conditions, as any excitement causes a disturbance of pulse and respiration. Particulars as to the date, mode of onset, and general character of the indisposition should be ascertained, but too much reliance must not be placed on the history. The general aspect of the child, the color of its face, the existence or not of restlessness or great depression, and the presence or absence of dyspnœa, must be noted. Some insight into the nature of the dyspnœa, if present, may be obtained. Thus, the inspiration may be attended by laryngeal stridor, or the voice may be hoarse; or, again, it may be noticed that the child breathes through the mouth—and this, if respiration be not greatly hurried, points to obstruction in the nose or naso-pharynx. An infant should be put to the breast in order to observe whether it is able to suckle without the frequent pauses which nasal obstruction produces.

The diagnosis of disease of the respiratory organs in the infant and child must be based almost entirely on physical examination; this, however, it is not generally possible to conduct in the methodical manner which may be followed in the adult. A young child may permit itself to be looked at, but unless unusually well and good tempered, or very ill, it is apt to resent auscultation a good deal, and palpation and percussion even more. In older children—say over two years old—the examination is best conducted with the child sitting on a high stool—if with a revolving top, so much the better. Unless the child be greatly exhausted, or show a natural disposition to lie down—and it is remarkable how acutely ill a child may be, and yet prefer to sit up or recline in its mother's lap—it is better not to have it in a recumbent attitude when examining the chest.

Inspection of the chest may afford much valuable information. The rickety rosary will indicate undue softness of the thoracic walls, and afford an important element in prognosis. Recession at the bases, particularly in front, and in the suprasternal notch, will indicate that air is entering the lungs with difficulty. The degree to which respiration, in the healthy child mainly abdominal, has been reinforced by thoracic movement or by the action of the accessory muscles will be observed, and any inequality in the expansion of the two sides can also frequently be noted. Some opinion also can commonly be formed as to the respiratory rate. This, in health, is faster in the infant and child than in the adult; and is in inverse ratio to the age of the child. The new-born infant makes from thirty to fifty respirations a minute, at one year the rate has fallen to twenty-five or thirty-five. It is somewhat slower during sleep, is easily altered by various circumstances, and is frequently somewhat irregular, with comparatively long pauses. If the respiratory organs be diseased, the breathing is commonly increased in rapidity, is regular and comparatively easy to count. The existence of distension, flatulent or otherwise, of the abdomen may also be observed.

Auscultation is, as a rule, best performed next in order. It should be conducted rapidly, and care should be taken that the hands are warm and soft. Though many still use the wooden stethoscope for one ear, preferring it on the ground that its use is attended by fewer adventitious sounds, it is better to become accustomed to the use of the binaural stethoscope, since the chest of a child can be examined with it much more rapidly, and the risk of hurting it by undue pressure is much less. A little practice in the examination of the chest in healthy infants and children will quickly train the ear to disregard the adventitious sounds, which, moreover, are really fewer than with the wooden stethoscope. The fingers brought into contact with the chest gather a certain amount of information as to its elasticity, and as to the existence of fremitus or tenderness, while the physician sees

precisely the area over which the sounds he hears are present. In examining the chest of an infant it is best to commence with the front, the patient lying in an easy attitude on its mother's lap. At the same time one axilla may be auscultated. Next, the infant should be placed on the upper part of the nurse's chest, with its hands and head resting lightly on her shoulder, and its buttocks supported by her hands. In this way the back is thoroughly accessible, and can be rapidly auscultated, as can also the other axilla. In children one year old and upward it is best to begin with the back, the child being in the sitting posture, and to examine in succession the supraspinous and interscapular areas, at the angles of the scapula, and the base. In the earliest weeks of life the breath sounds are weak, but thereafter become gradually louder and harsher; so that at about six months of age the vesicular murmur is louder, higher pitched, and rougher, almost blowing, and expiration may be distinctly audible—the condition to which in the adult the term "puerile breathing" is applied. Each axilla may be auscultated from below upward, and finally the front of the chest from above downward.

Palpation may then be rapidly completed by placing the hands on the two sides to detect any inequality of expansion; to estimate the heat of the skin; and to complete observations as to the existence of rickety deformities or undue softness. The position of the apex-beat should also be ascertained.

Percussion should be lightly performed. The younger the child, the less the importance to be attached to variations in the percussion note, unless the alteration be very marked. In a healthy infant, breathing calmly, the percussion note is almost tympanitic. But when it is beginning to cry, the abdominal pressure forces up the viscera, the liver in particular, and the note becomes somewhat dull over the left base, and flat and short over the right. Percussion over the upper parts of the chest, especially in front, will, if too forcible, produce a "cracked-pot" sound.

The *pulse* in early infancy is rapid—120 to 150—and easily quickened. It is difficult to count it at the wrist, but the rate and general character can be ascertained by auscultation. The rate and force of the heart are easily disturbed, so that not much information of general clinical value can be obtained from its examination. The first sound is short, and toneless as compared with that of the adult, and the second less sharp, owing, probably, to the low arterial tension characteristic of infancy. During the second year of life the pulse still is fast, over 100, but becomes slower in the third year, and falls to the adult average at about the seventh year, by which age also the sounds have assumed their characteristic qualities. Pathological slowing of the pulse is not common in infancy and early childhood, and when observed is usually associated with tuberculous meningitis or jaundice.

The *abdomen* in the infant is larger in proportion to the rest of the body than in the adult, and owing to the small size of the pelvis is rendered more protuberant. This protuberance, however, is towards the front, and the sides of the belly should not be visible when the child is regarded directly from the back. In health it is firm and uniform to the touch, and if the infant is in a good temper, to begin with gentle manipulation appears to give it pleasurable sensations. Neither the liver nor the spleen can be perceived with any confidence. The liver occupies nearly half of the abdominal cavity; its lower border reaches from the left hypochondrium across the epigastrium almost horizontally to the right hypochondrium, descending in the flank a little lower on the right than on the left. Emaciation, or laxness of the abdominal walls due to past distension, renders it easy to palpate the lower border. Under similar circumstances the spleen when enlarged is easily felt, usually best by slipping the pulps of the fingers obliquely over the edge of the thorax, and carrying them downwards; if the one hand be placed under the flank and the other used for palpation from the left side, the spleen may easily be pushed out of the way, and missed even when moderately enlarged. The movements of the abdomen in respiration should be free, and their absence points to serious abdominal disorder, and probably to involvement of the peritoneum. The existence of gurgling in the intestines and of enlarged glands will also be ascertained during palpation. Great flatulent distension of the intestines renders the belly more or less globular, tense, and tympanitic on percussion. In chronic gastro-enteritis the lower part of the belly feels doughy; while in the upper part there is often tension, and a tympanitic percussion note owing to flatulent distension of the stomach and colon. Marked retraction of the belly combined with softness to the touch will suggest tuberculous meningitis (*q. v.*).

Examination of the *head* will show the condition of ossification, and the presence or absence of cranio-tabes. The condition of the anterior fontanelle, whether tense or retracted, will afford information as to the state of the circulation, which is often more valuable than that given by the pulse or heart sounds.

Retraction of the Head.—In infants and young children the first symptom of meningitis to attract attention may be retraction of the head and rigidity of the muscles at the back of the neck. This is due to meningitis of the posterior fossa; if the inflammation extend into the spinal canal the tonic rigidity involves also the muscles of the back. The cause of the inflammation is not always to be ascertained. In some cases it is tuberculous, and the more general symptoms of tuberculous meningitis follow. Cases running a very chronic course have been attributed to syphilis. Retraction of the head, dating from birth, is attributed by Gowers to meningeal hæmorrhage in the neighborhood

of the medulla, or to laceration of the cerebellum. Tumor in this region may also cause retraction. Instances of retraction of the head in infants, slight in degree and short in duration, are sometimes met with, and are apparently functional; such children sometimes present symptoms of tetany (*q. v.*). Pneumonia of the apex is in many cases accompanied during the stage of onset by more or less marked retraction of the head. Rheumatism of the muscles of the neck and back, and acute or sub-acute cervical adenitis are among local causes of retraction. Middle ear disease and certain peripheral irritations, especially gastro-intestinal disturbance, and infestations by intestinal worms (ascaris), may also determine retraction, though probably such cases should be classed as examples of tetany. The retraction may be slight and intermittent, or extreme and constant, so that the occiput is in contact with the back. The infant lies on its side if in a cradle, but prefers to be nursed, the mother's arm supporting the head. When put into the sitting posture the retraction becomes greater, and the infant evidently suffers pain.

Fever.—The main sources of the body heat are the muscles and the abdominal organs; the main sources of loss the skin and lungs, mainly the former. Fever is produced by toxæmia, the poisonous substances acting, possibly directly on the metabolic processes of the tissues, and certainly indirectly by disturbing the heat-regulating nervous mechanism, which presides over both the production and the loss of heat. As Broadbent has well said, the fact "that febrile heat is not vague and irregular, but that there is the substitution of a morbid for a normal balance, is evidence of nervous control." During the febrile process there is increased loss of carbonic acid by the lungs and of nitrogen by the urine. At the same time, there is an arrest of the digestive secretions, so that with increased destruction and diminished assimilation there is necessarily a more or less rapid wasting of the tissues and diminution of their functional activity.

The temperature in childhood is easily affected. Slight disturbance may cause it to rise above the normal, and the height to which it may be raised may be out of proportion to the severity of the pathological process. The converse proposition that the temperature in childhood is easily reduced when abnormally high is true also as a general statement. It would, however, be a mistake to regard lightly the presence of high temperature in childhood. A single observation may have little significance, but if the pyrexia continue it is as definite an indication of the existence of disease as in the adult. On the whole, the greater the care with which patients are examined the more rarely will paradoxical temperatures be met with. On the other hand, it must be remembered that causes which in the adult would lead to a rise to perhaps 100° F. will, in infants and young children, produce temperatures of 103° to 105° F. This is more

especially true in its application to children of excitable temperament.

Subnormal temperature, when observed in childhood, is usually a symptom of marasmus, and is a bad omen as a sign of great nervous exhaustion. Under careful treatment, however, infants may survive temperatures as low as 96° F., or even lower.

Fever, to whatever cause it may be due, renders the patient specially liable to various secondary affections; in particular, to bronchopneumonia and to gastro-enteritis.

CHAPTER III.

DISEASES INCIDENTAL TO BIRTH.

Hæmorrhagic Extravasations during Parturition; Meningeal Hæmorrhage—Icterus Neonatorum—Acute Fatty Degeneration of the New-Born—Acute Hæmoglobinuria of the New-Born—Mastitis—Erysipelas Neonatorum—Erythema Neonatorum—Diseases of the Navel—Tetanus Neonatorum—Sclerema Neonatorum—Œdema Neonatorum—Melæna Neonatorum—Pemphigus Neonatorum—[Hæmorrhagic Diseases of the New-Born].

Hæmorrhagic Extravasations During Parturition.—During the act of parturition hæmorrhage may occur into the skin, subcutaneous tissues, muscles, or viscera of the infant.

The causes are to be sought (1) in the great delicacy of the vessels; (2) in the force exerted by the uterus on the child, which may be compressed strongly against the maternal parts, while the blood may be squeezed mechanically into certain organs; and (3) in pressure or traction exerted by the hand of the obstetrician or by forceps. Asphyxia, which is capable of producing sub-serous petechiæ, will have the effect of reinforcing other causes tending to produce hæmorrhage into the substance of organs.

Cephalhæmatoma is the term applied to the effusion of blood which often takes place between the skull bones and their periosteum. The bone most often affected is the right parietal, next to that the left, more rarely the occipital, frontal, or temporal. The hæmorrhage is limited by the attachment of the periosteum at the sutures, but both parietal bones may present blood tumors. The swelling continues to increase for some days after birth. It is soft and fluctuating, and by deep pressure the underlying bone may be felt. After a time the edge becomes hard, and eventually the periosteum forms a ring of bone all around the hæmatoma. Plates of bone may also form in the periosteum over the fluid, and give a crackling sensation when the swelling is handled. The blood is absorbed in the course of a few weeks, but the ring of bone persists much longer, often for many months. Occasionally the external effusion is associated with hæmorrhage between the skull and the dura mater, and a connection may exist between the two collections.

The diagnosis is usually easy. A question hardly arises until after the time at which a caput succedaneum would have disappeared.

Cephalhæmatoma is, in fact, distinguished from all other conditions with which it might be confused, with the single exception of meningocele (or encephalocele), by the date of its appearance and the existence of fluctuation. Meningocele, however, corresponds in situation with a fontanelle or suture, pulsates, and becomes more tense when the child cries. Moreover, the aperture through which it protudes can be made out, and ought not to be confused with the bony ring around a cephalhæmatoma; moreover, in the latter condition the underlying bone can be felt. The **prognosis** is good unless symptoms exist pointing to the concurrence of intracranial hæmorrhage.

The treatment should consist in protecting the swelling from injury. Incision is unnecessary, and no local or internal medication is known which will hasten the disappearance of the effused blood. This will take place in time, and the bony ridge will gradually disappear also.

Meningeal Hæmorrhage is the most important of the extravasations which attend birth, owing to the fact that it produces serious permanent symptoms should the child survive. Compression of the skull during parturition may cause congestion and œdema of the cerebral meninges and of the brain substance, with or without hæmorrhage into or beneath the membrane. Hæmorrhage between the skull and dura mater occurs in association with fracture. Hæmorrhage into the pia mater or arachnoid is the most frequent lesion in infants dying in consequence of injury during parturition. In most cases blood is effused over the convexity on both sides and at the base, sometimes on one side only. In rare cases, hæmorrhage takes place into the ventricles or choroid plexus, or into the substance of the brain. Judging from the conditions found in still-born infants, intracranial hæmorrhage occurs more frequently in those delivered by the forceps[1] than in those born by the breech, and in the latter more frequently than in those born naturally by the head. It may occur during rapid delivery as well as slow, in multiparæ as well as in primiparæ, in small as well as in large children.

The lesions of the *spinal cord* found in still-born children are congestion of the whole, or of the anterior cornua, or of the surface, and hæmorrhage outside the theca into the meninges, or into the cord (especially the anterior cornua). The lesions of the *abdominal organs*, which may be produced during birth, may be enumerated as follows: *Liver:* Congestion of the substance, hæmorrhage at the surface. *Kidneys:* Congestion with or without hæmorrhage into the

[1] Herbert Spencer, to whose article (*Trans. Obstet. Soc.*, vol. xxxiii.) I am much indebted, makes the remarkable statement that "cerebral hæmorrhage was found in every case in which the forceps was employed to deliver living children who died during or shortly after birth."

hilum, beneath the capsule or into the substance. Hæmorrhage into the pyramidal portion may be a cause of suppression of urine and death a few days after birth. *Suprarenal capsules:* Congestion with or without hæmorrhage. *Spleen:* Congestion, hæmorrhage (rare). *Intestines:* Contain blood occasionally; the *stomach* more rarely, and then derived from elsewhere. In the thoracic organs the *lungs* may show sub-pleural petechiæ, or more massive hæmorrhages into the substance, especially at the base. In the *heart* there may be small hæmorrhages beneath the pericardium and into the valves. Extensive extravasation may take place into the *parotid gland*, and Spencer suggests that the pressure thus exerted on the trunk of the facial nerve may be one of the causes of facial paralysis in the new-born.

Hæmorrhage may occur also into *muscles* during delivery. Of these accidents the most important is *hæmatoma of the sternomastoid*, since it is a cause of wry-neck which may last for months, and is, in some cases, possibly a large proportion, permanent. It is due, usually, to great stretching of the muscle during delivery of the after-coming head. Less often it is caused by pressure of one blade of the forceps. It occurs also, occasionally, in vertex delivery. It is generally noticed first a few weeks after birth, when a small rounded or oval tumor is found in the muscle, generally in its upper part and on the right side. Sometimes, however, the first symptom which attracts attention is that the neck is not held straight. At a later stage the swelling is replaced by a sclerosis of the muscle, which is shortened and feels like a tendinous band under the skin. Petersen has suggested that in some, if not all cases, there is a congenital defect in the development of the sternomastoid, which is shorter than natural, and, therefore, more easily injured. This suggestion finds support in the observation that in many cases of congenital wry-neck the development of the whole of the face on the affected side is defective, so that it appears atrophied, as compared with the other.

The **skin** of a healthy infant, twenty-four hours old, when, that is to say, the congestion which so frequently attends birth has passed off, is of an almost uniform deep pink or red color. This is due to hyperæmia, attended, perhaps, by some effusion of the coloring matter of the blood. As a rule, the red coloration disappears in about a week, when the skin assumes the natural "flesh tint," but in some cases the red color is succeeded by a distinct and almost universal yellow tint. To this condition is applied the term

Icterus Neonatorum.—Since this occurs very frequently, and is commonly unattended by any other obvious departure from health, it has been thought by some to be physiological. It occurs, however, more often among weakly children, those born prematurely, or in cases in which, during parturition the umbilical cord has been compressed or torn. It occurs also in association with exposure to

cold, with atelectasis pulmonum, and with imperfect establishment of respiration. It is met with more frequently in lying-in and foundling institutions than in private habitations.

The pathology of icterus neonatorum has given rise to much controversy. *Post-mortem* the serous membranes, the endocardium, the intima of the arteries, the liver, spleen, and kidneys, and the brain, have a yellow color which, according to Orth, is due to the presence of bilirubin. Uric acid infarcts when present in the kidneys are deeply pigmented, and the urine contains yellow bodies, which Cruse has shown consist of bile pigments, either free or in epithelium cells or hyaline cylinders. In some cases a plug of mucus has been found in the ductus choledochus. All these facts point to the liver as the source of the pigment. On the other hand it is urged that since the fæces have the normal yellow or brown color, and as the urine does not contain much bile pigment, if any, and as the ductus choledochus is commonly found patent after death, the icterus must be hæmatogenous. In support of this theory is pointed out that during the first few days of extra-uterine life a great destruction of red blood-corpuscles takes place, by which much pigment is set free, while, at the same time, the metabolism of albumen is very active, so that great calls are made upon the functional activity of the liver. The most acceptable theory appears to be that the jaundice is due to a temporary hepatic insufficiency brought about in the manner indicated. This would produce a more marked effect if the ductus choledochus were blocked by a mucous plug, as it is in some, at least, of the fatal cases ; under such circumstances, the retention of color by the fæces must be attributed to the meconium remaining in the intestines.

The characteristic **symptom** is the yellow tinge of the integuments, generally most marked on the face and chest, of the conjunctivæ, and of the gums, as can be made evident by pressing gently with the finger. The yellow tint begins to be noticeable about the second or third day of life ; if the skin still retains much red coloration, it may be brought out by pressure with the finger, the resulting patch of temporary anæmia having a yellow tinge. The child is not ill, suckles well, and the pulse is not slow. The urine is clear, generally of a light color, and contains a large quantity of urea and uric acid. The fæces are yellow or brown, and soft.

The diagnosis must rest upon a general consideration of the circumstances of the case, and especially on the time of the onset of the icterus. Congenital icterus points to a serious condition, to severe syphilis, or to congenital deficiency of the bile ducts, or occlusion of the ductus choledochus, or of the duodenum. The association of jaundice with umbilical inflammation is of serious significance. The occurrence of jaundice as a symptom of acute fatty degeneration, and with acute hæmoglobinuria, will be mentioned later.

The prognosis is good in uncomplicated icterus neonatorum, though the fact that a large proportion of the children thus affected are weakly, and very liable to suffer from gastric catarrh, must be borne in mind.

The treatment should consist in keeping the child warm, giving it fresh air, and feeding it carefully and regularly with, if possible, its mother's milk. Mercurial and other laxative drugs should be avoided.

Acute Fatty Degeneration of the New-Born is a rare and fatal form of disease observed in new-born infants. It is characterized by a parenchymatous inflammation of the viscera and of the skin, accompanied by hæmorrhages and followed by fatty degeneration.

The etiology of the disease is obscure; it is probably an infective process, and may perhaps best be regarded as a form of septicæmia. In some cases there is obvious disease of the navel, which may then reasonably be regarded as the point of entry of the infection. The disease has been observed most often in infants which have been asphyxiated at birth, but is by no means confined to the weakly.

The **symptoms** are progressive. In most cases respiration is never properly established, and the face, and to some extent the skin generally, is cyanosed. The cyanosis, as a rule, deepens gradually, but in some cases suddenly, and eventually gives place to an icteric tint. The subcutaneous tissue may become œdematous. Ecchymoses may appear on the skin and mucous membranes, and hæmorrhage may take place from the navel. Vomiting is a common symptom and the rejected matter is blood-stained; the stools contain blood, as does also the urine. The infant presently becomes collapsed, and death usually ensues in the first or, at latest, the second week. After death hæmorrhages will be found to have taken place into the serous and mucous membranes, and there is fatty degeneration of the liver cells, the myocardium, the renal and pulmonary epithelium, and the intestinal villi. Hæmorrhagic infarctions also may be found in the lungs, and hæmorrhages into the stomach, intestines, and navel.

The **prognosis** in a well-marked case in which the diagnosis can be definitely made is exceedingly bad.

The **diagnosis** is often difficult, and in a case with a rapid course, especially if seen only shortly before or after death, it may be difficult to exclude poisoning by phosphorus or arsenic except by chemical examination. The resemblance of the body after death to that of a child killed by suffocation may be close, but the discovery of extensive fatty degeneration of the viscera will indicate the true cause of death. The possibility that the disease is septicæmic in nature has been mentioned and when the navel is diseased it will be difficult to exclude ordinary septicæmia from that source.

The **treatment** can only be symptomatic. The partial asphyxia may be combated by artificial respiration, and probably the use of

oxygen might be of benefit. Hæmorrhages must be controlled by ordinary means, and special attention should be given to feeding the infant at regular intervals, giving by preference the mother's milk, which must be drawn off if necessary.

Acute Hæmoglobinuria of the New-born (*Winckel's Disease*) is a rare and very serious general disease, probably of infective nature. It is characterized by cyanosis and hæmoglobinæmia and hæmoglobinuria.

The disease attacks infants, who often appear to be robust, about the fourth day of life. The infant becomes restless and refuses food. The skin assumes a yellow or greenish tint, the respiration is hurried, but the pulse is not quickened nor the temperature raised. The urine, which is clear and of a brownish or olive-green color, contains epithelial cells, hyaline cylinders, masses of detritus and hæmoglobin, but no blood-cells. Vomiting and diarrhœa are not infrequent, and convulsions sometimes precede death, which is almost invariably the termination of this disease.

The pathological conditions found after death point to the infective nature of the morbid process. The kidneys are large and dark, with small hæmorrhages in the cortex, and hæmoglobin infarcts in the pyramidal portions. All the viscera are hyperæmic, and have a yellowish tinge, and all, but especially the serous membranes, show punctiform hæmorrhages. The spleen is large, firm, dark, and greasy on section. There is fatty degeneration of the liver and extensive desquamation of the intestinal epithelium, with swelling of Peyer's patches. The blood is dark with a greenish tinge and contains an excess of white cells and red cells much altered, some nucleated and others degenerating.

Mastitis.—In the healthy infant of either sex the mammary glands on or about the fourth day of life begin to secrete a small quantity of fluid, which has the chemical and microscopic characters of milk, and contains colustrum corpuscles. The glands enlarge during the four or five following days, and then gradually decrease in size, until at the end of the third week, as a rule, they cease to be conspicuous, and the secretion is arrested. The enlarged mamma is a firm conical body, an inch or less in diameter, which is freely movable but a little tender. A drop of opalescent milk can be squeezed out as a rule, but usually the swelling is inconspicuous and passes unobserved. Occasionally the enlargement attains greater proportions, one or both glands become hard and very tender, and the overlying skin is reddened; in fact, a condition of mastitis is established which may run on to abscess.

The enlargement and functional activity of the mammæ in newborn infants is a physiological process, and it is very possible that the occurrence of mastitis neonatorum is to be attributed in most in-

stances to the superstition which leads a nurse to manipulate the glands rather violently to "break the nipple-strings," or to draw off "the witches' milk." The inflammation is associated with the presence of pyogenic cocci, and may be attended by a good deal of fever, restlessness, and loss of appetite. Under suitable treatment it usually subsides without the formation of an abscess. When an abscess froms it almost invariably heals readily after incision, but in weakly children may cause some trouble, and has been known to burrow under the pectoralis, and to cause extensive sloughing of the skin.

Treatment should be directed, in the first place, to the prevention of mastitis by protecting the glands from injury. When they become enlarged and tender they should be smeared with boracic ointment and covered with pads of cotton-wool bandaged lightly on. If their increased size, tenderness, and the redness of the skin indicate that inflammation is commencing, the ointment should be replaced by extract of belladonna and glycerine (equal parts). Hot fomentations or poultices may be used at once, or after failure of the belladonna. Not uncommonly a purulent fluid eventually exudes from the nipple, and the mastitis subsides without the necessity for incision. If fluctuation can be made out, it is best to make an incision radiating from the nipple, and as the segments of the gland are sometimes affected successively, it may be necessary to make more than one opening.

The **diagnosis** is easy from the physical signs, but it is necessary to remember that some enlargement and tenderness of the glands is a physiological process.

The **prognosis** is good, though in a marasmic infant the pain, restlessness, and fever attending mastitis may aggravate the condition seriously. Severe mastitis with abscess in infancy has been followed, in some cases, by imperfect development of the gland in girls at puberty.

Erysipelas Neonatorum.—The new-born infant is liable to suffer from erysipelas in two clinical forms:[1] (1) an acute general infection and (2) a creeping cutaneous affection spreading from some skin lesion.

1. The *acute general disease* occurs, as a rule, in association with puerperal fever in the mother, or in institutions. The onset of the erysipelas is sudden, and is accompanied by severe general symptoms, high temperature (105° F.), vomiting, diarrhœa, and jaundice, and is complicated frequently by pleurisy, peritonitis, and arthritis. Convulsions ensue, the infant passes into a condition of stupor, and life is seldom prolonged beyond the second day of illness.

[1] See also Sclerema neonatorum, p. 48.

2. Creeping erysipelas starts from some lesion of the skin—from the navel, the penis after circumcision, or a patch of intertrigo. It varies very greatly in severity, but is always a serious disorder. It is not always possible to trace infection from a previous case, though the disease used to prevail as an epidemic in lying-in institutions before strict antiseptic precautions became the rule. It is now seen most often in the infants of the poor living in insanitary surroundings, and the victims are often fat and strong looking. In some cases the infection appears to be conveyed by the napkins, and the area of affected skin is sharply defined by the edges of these cloths. Once begun, the process tends to spread to the whole cutaneous surface, sometimes very rapidly, but more usually slowly, so that the parts earliest affected are recovering while others are being invaded. After the red color has faded from the skin, a good deal of soft œdema may remain. In the loose parts, as, for instance, the scrotum, the swelling may be very great. More or less extensive areas may become the seat of phlegmonous inflammation, and abscesses may form, or necrosis of skin may occur. The general symptoms vary a good deal in severity. There is, usually, continuous fever, with morning remissions, but in the more chronic cases, especially in weakly infants, the temperature may be little above the normal, and may even be sub-normal in the morning. The appetite is sometimes retained. The pulse becomes small, rapid, and weak. The disease may be complicated by diarrhœa and vomiting, by pneumonia, or by peritonitis. The advancing border of cutaneous infiltration is generally very regular and well-defined, and upon the inflamed skin are seated, in many cases, small vesicles containing a clear white or yellowish fluid. The **prognosis** is bad, although recovery sometimes takes place in the less acute cases. **Treatment** exercises little influence on the course of the malady. The infant should be fed with its mother's milk in small quantities at short intervals, and alcoholic stimulants prescribed, a teaspoonful of good claret or champagne, or ten drops of brandy in water every two hours. The internal administration of perchloride of iron is well borne, and may be of service (liquor ferri perchloridi, ♏ ij–iv, every two hours alternately with the stimulant). An attempt may be made to check the spread by painting the edge with absolute alcohol or with silver nitrate (mitigated stick, or a solution gr. xx to ℥j).

The **diagnosis** is not difficult as a rule. In the less acute cases, in which hesitation is most likely to be felt, the degree of cutaneous infiltration, the well-defined regular slowly-spreading edge, the fever, and the general depression usually leave little doubt as to the nature of the disease. In syphilitic infants the skin affection about the buttock may, when it is beginning, be attended by extensive redness and infiltration of the skin, corresponding more or less closely with the

area covered by the napkin, but this is seldom seen during the first two weeks of life. The condition with which creeping erysipelas is most liable to be confounded is

Erythema Neonatorum.—This is a mild affection, though the erythema may be exceedingly widespread. It makes its appearance usually about the second or third day of life, and spreads rapidly over the greater part of the trunk and limbs. The skin is red, full, and a little tense. The infant is restless, and loses appetite, but the temperature is little, if at all, raised. After a day or two the erythema begins to fade, and there is usually a little fine desquamation. The **diagnosis** from erysipelas and scarlet fever must be made from a careful examination of the characters of the rash, the mild nature of the general symptoms, and the absence of sore throat, or much furring of the tongue. The **treatment** should consist merely in keeping the parts powdered, and in the use of lukewarm baths, which diminish the restlessness.

Diseases of the Navel.—The umbilical cord usually separates spontaneously about the fifth day without giving rise to any trouble, but occasionally the navel becomes the seat of inflammatory or other morbid processes, of which the most common is

Ulceration.—After the separation of the cord, a small granulating discharging surface is left, which often becomes covered with a crust. In some cases the granulations are so exuberant that they form a small tumor of irregular shape, which projects from the navel. To this condition the term *fungus umbilicalis* has been applied. This granuloma has a smooth moist surface which bleeds easily, and the puriform discharge from it irritates the surrounding skin, which becomes red and excoriated. The *treatment* of umbilical ulceration should consist in antiseptic applications—boric acid lotion followed by the application of boric ointment, or mild white precipitate ointment, and the use of an antiseptic dusting-powder. Fungating granulations should be touched with lunar caustic stick, and dressed with one of the ointments already mentioned. Neither condition is in itself of any serious consequence, but they draw importance from the fact that the ulcerated surface may serve as the point of entrance of erysipelatous or other infection.

Phlegmonous Inflammation of the cutaneous structures about the navel may ensue upon ulceration, or after the normal detachment of the cord. A conical, red, tender swelling like a huge boil, with the navel more or less everted at its centre, forms, and suppuration generally ensues, the resulting abscess, if not incised, opening either at the navel or in its neighborhood. Sometimes the pus tracks downwards, and eventually a long sinus forms which may reach to the pubes and prove very troublesome. The patient during the attack suffers from pain, restlessness, want of appetite, and slight fever.

If a sinus form the infant becomes much exhausted by the discharge, which is often copious, and may suffer much from dermatitis set up by it. In the *treatment* these risks must be borne in mind, and if poultices, or hot fomentations, with, perhaps, the addition of belladonna and glycerine, fail to arrest the inflammation, an incision should be made near the umbilicus as soon as fluctuation can be made out, the abscess cavity thoroughly drained, and all discharges taken up by a pad of absorbent cotton-wool frequently renewed, an antiseptic ointment being applied to the surrounding skin. A possible but, happily, rare complication of phlegmonous inflammation of the navel is *gangrene* of the skin over the inflamed area. The whole thickness of the abdominal walls may be involved, laying bare the peritoneum, or producing an opening into the peritoneal cavity, through which the intestines, glued together by inflammation, are visible. Gangrene is attended by extensive surrounding inflammation and œdema of the skin, by fever, and by rapid loss of strength. The *treatment* should consist in careful systematic feeding and the use of stimulants; locally, hot antiseptic fomentations should be used to encourage the separation of the sphacelus; and, later, antiseptic powders or iodoform may be freely dusted on, the wound being thoroughly irrigated every three or four hours with boric-acid solution, and again dusted with iodoform.

Thrombosis of the umbilical vessels is another complication of ulceration of the navel. The arteries are affected more often than the vein. The infective agent is usually the streptococcus pyogenes. *Post mortem* the affected arteries are hard and thick, of a brownish color, and surrounded by gelatinous œdematous tissue; they contain, according to the age of the arteritis, a soft reddish thrombus, or puriform material produced by its breaking down. The coats of the vessels are infiltrated and may eventually give way. When there is phlebitis the vein is filled with breaking down thrombus, its walls are infiltrated, and the surrounding tissue œdematous. The ulceration of the navel may have healed. Evidences of embolism may be found in many of the internal organs—the brain, spinal cord, lungs, kidneys, liver, spleen. Pneumonia, purulent pleurisy, and joint affections are not uncommon; in fact, every lesion of acute pyæmia may be met with.

The general *symptoms* are not very characteristic. The infant becomes restless, refuses the breast, is feverish, and soon becomes jaundiced. It lies upon its back, with the knees drawn up, and it may be possible to feel the two thrombosed arteries as hard cords running down from the umbilicus on either side of the linea alba; if there be phlebitis there will be some swelling and tenderness in the middle line above the umbilicus. The *prognosis* is very bad, especially in premature children, and death frequently ensues in a

few days. In other cases the infant may survive for three weeks or more, evidence of the pyæmic infection of various organs being afforded from time to time. For the *prevention* of this very fatal disease we must look to antiseptic treatment of the navel, and to the separation of new-born infants from persons suffering from erysipelas or puerperal fever. The *treatment* is unsatisfactory, since direct applications to the thrombosed vessels appear to be impossible. If any indication for surgical interference is afforded it ought to be followed without delay, as a chief danger is the occurrence of septic embolism. The restlessness by which the infant's strength is exhausted may be relieved by warm baths or by warm packs, and in addition to regular feeding with its mother's milk, wine or brandy may be given, and the effect of quinine tried in increasing doses.

Hæmorrhage from the umbilicus may come, (1) from the *arteries*, owing to injury during birth, or to the cord being insufficiently secured, or, at a later date, owing to gangrene of the cord before the vessels have become occluded. In weakly infants, in whom the pulmonary circulation has been imperfectly established, a considerable amount of blood may be lost in this way, but the hæmorrhage is, as a rule, easily checked by a pad of cotton-wool, or by applying a fresh ligature to the cord, though the accident is most likely to occur when the first has been applied too near the navel. Hæmorrhage after gangrene of the cord must usually be treated by ligature over pins. (2) The bleeding may be an *oozing* from the navel, after the separation of the cord, and may be an indication of a general disease, hæmophilia, syphilis, septicæmia, or acute fatty degeneration. The bleeding begins usually about the fifth day of life, and before the cord has separated completely. It is not arrested by compression and there are simultaneons hæmorrhages into the internal organs, and into the skin. The **prognosis**, owing to the fact that the bleeding depends upon a general condition, is bad, and life is seldom prolonged for more than a few days. [We must, however, distinguish between umbilical hæmorrhages due to these general causes, and those of an infectious origin occurring as a manifestation of the disease known as the *Hæmorrhagic Disease of the New-born*, described more fully below.] This form of umbilical hæmorrhage is rare, and the treatment is unsatisfactory. Styptics should be used, and Wright's physiological styptic[1] suggests itself as well adapted to the purpose. If these fail, the navel must be ligatured with a thread carried round it on a needle. The child must be kept quiet, fed carefully, and the general condition treated.

Tetanus Neonatorum is the same disease, due to the same infective agent—the tetanus bacillus—as that which occurs in adults. The chief peculiarity is that, as a rule, it at first affects the muscles

[1] *Lancet*, 1883, i., p. 435.

of the jaws and face, and has hence been known as trismus neonatorum.

Etiology.—The infection finds entrance by the navel, and negligent or dirty methods of treating the cord are the chief contributory causes. The disease has prevailed as an endemic in certain localities (Faroe Islands), and in institutions, and want of ventilation has appeared to be a determining cause in such circumstances. Exposure to cold and the use of too hot baths have also been considered to be among the remote causes. In some instances the infection appears to have been carried by midwives.

The **symptoms** begin usually between the fifth and the ninth days of life, and the first thing to attract attention is that the infant has difficulty in suckling, and that the attempt is accompanied by contraction of the masseters and the orbicularis oris. These muscles are found to be hard to the touch. The infant is restless, cries much, and frequently wakes from sleep with a cry. The muscular cramps are brought on by any movement; they involve gradually a larger number of muscles, and finally the attacks come on without obvious cause. The brows are wrinkled, the eyes closed, the face drawn, the alæ nasi dilated. Next the head and neck become stiff in each attack, and in the intervals relaxation is incomplete. The tonic contractions finally involve the trunk muscles, and in the attacks there is well-marked, often extreme opisthotonos, the abdomen is hard, the hands clenched, the legs abducted. During the attacks respiration is arrested and the surface becomes cyanosed; in the intervals it is shallow and irregular. The pulse is fast and thready. The temperature may be little raised at any period of the disease; on the other hand, it may mount gradually from the beginning until it attains 104° or 105° F. shortly before death, or it may be high from the first. The urine contains albumen and casts. The usual termination is in death, which may occur within twenty-four hours, though life may be prolonged for a week or ten days.

The **prognosis** is extremely unfavorable. A low temperature is of good augury, and the first signs of improvement are a lengthening of the interval between the attacks and more complete relaxation. Improvement in any case is very gradual, and recovery can only be looked for if the infant has a considerable reserve of strength. **Treatment** has little effect in the more acute cases, but in the less severe, advantage may be hoped from the systematic use of chloral hydrate ($\frac{3}{4}$ to 1 gr. every hour). If swallowing is impossible, the remedy must be given by enema (gr. $1\frac{1}{2}$). Momentary relief during the attacks may be obtained from inhalations of chloroform. With the chloral may be combined potassium bromide, 15 to 20 grains being given in the course of the day. Sulphonal (gr. j–ij) every three or four hours by enema has also been recommended, and extract

of calabar bean administered by hypodermic injection (gr. $\frac{1}{2}$ dissolved in ♏x of water) has given good results in some hands. The infant should be kept very quiet in a shaded room. Its food should consist of its mother's milk and should be given regularly, as the main hope of recovery, even in the least severe cases, is in maintaining sufficient strength to enable the infant to outlive the disease. Rectal alimentation is recommended, but in some cases feeding with the nasal tube seems to afford promise of better results. If the navel be ulcerated or otherwise inflamed, it should be thoroughly treated with antiseptics. The use of tetanus antitoxin has not been followed by constant or obviously beneficial results, but it does not seem to be attended by any inconveniences, and may therefore properly be resorted to.

Sclerema Neonatorum is a rare disease characterized by a peculiar hardening of the cutaneous structures. It begins usually in the calves, but sometimes in the cheeks, during the first week of life. The subjects are usually premature or weakly, and the majority of cases have been observed in institutions, but beyond these facts nothing is known as to the etiology of the disease. The **morbid anatomy** does not throw much light on the pathology. There is an overgrowth of the connective tissue of the skin and subcutaneous layer, with an absorption of fat, and a marked dryness. The viscera show no morbid changes beyond pulmonary collapse, which is more probably a consequence than a cause of the sclerema.

The **symptoms** are characteristic, for the peculiar hardness of the skin resembles no other condition. It does not pit on pressure, and feels like leather or wood; the smaller folds are obliterated, the larger strongly marked with firm edges. Its color is at first whitish or marbled, but later there is a yellow tinge. The induration spreads with varying rapidity, and may eventually involve almost the whole skin, so that the infant lies in a condition resembling *rigor mortis*, the only movement perceptible being respiration; if lifted up it remains rigid, like a wooden doll. Rigidity of the lips and cheeks renders suckling difficult or impossible. Respiration becomes shallow and irregular, the pulse if it can be felt, which is often not possible owing to the hardening of the skin, is small, and the heart-sounds weak. There is constipation, and very little urine is passed. The temperature falls below normal, even to 85° F. or lower, and the mouth feels cold to the finger introduced into it. Death is due to progressive exhaustion, or not infrequently to intercurrent pneumonia.

The **prognosis** is very bad in all cases in which the disease has become extensive, but there is a condition which resembles sclerema, and is generally assumed to be identical, which is not very serious. It is generally confined to the buttocks, groins, and front of the abdomen, covering an area coinciding with that enveloped by the napkins. The skin becomes hard, solid, and does not pit on pressure;

the folds of the nates are closely opposed, and there may be difficulty in extending the thighs. The skin is not white or yellow but of a deep red, like raw ham; firm pressure with the finger produces little alteration in the color. The surface is glazed and dry. Sometimes a few other similar but smaller patches may be found on the arms or calves. In these cases the general health does not suffer, and after a week or two the thickening and redness begin to fade and finally disappear. Whether this condition, which, after developing rapidly in a few days, remains localized until its recession, should be regarded as pathologically identical with sclerema, is doubtful. Barrs, who has given a good description of a case,[1] holds that it should, and that true sclerema sometimes begins in this way. Clinically, at least, the two conditions differ in appearance and result. True sclerema is rare, but this local condition is not uncommon.

The **diagnosis** of sclerema, if the local condition just mentioned be excluded, is not difficult. It is distinguished from œdema neonatorum, with which it has been confused, by the absence of pitting and by the color of the skin. At the same time, it must be remembered that sclerema has, in a few cases, been preceded by some œdema. The history of the case, and a careful observation of the cause of the rigidity, ought to prevent any confusion with tetanus, and some help may be obtained from the temperature, which in tetanus, if not raised, is seldom below the normal as in sclerema.

The **treatment** of sclerema, owing to the absence of any certain knowledge as to its pathology, must be symptomatic. An attempt should be made to maintain the body temperature by placing the child in an incubator or "artificial nurse," if one be at hand, or by wrapping the body in cotton-wool and applying artificial warmth by means of hot bottles or sand bags under blankets in a bed. Stimulants—wine, brandy, ammonia, musk, camphor—should be given, and food, preferably the mother's milk, at regular intervals, in a spoon.

Œdema Neonatorum is not a disease but a symptom of various pathological conditions similar to those which produce œdema at other ages—heart disease (fœtal endocarditis) and nephritis. It occurs also in marasmic infants, especially in those affected by pulmonary atelectasis, and occasionally in congenital syphilis. It is sometimes a sequel of erysipelas, and occasionally precedes sclerema.

The **symptoms** present a general resemblance to sclerema, and the œdema may even be so tense and widespread as to interfere with movement. The skin is pale or marbled, and always pits on pressure. The œdema begins in the lower extremities, and gradually ascends; when it reaches the genital organs it often produces extreme distension and deformity. The general condition of the infant is

[1] *Brit. Med. Journ.*, vol. i., 1889, p. 994.

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usually one of great depression, and the temperature may be far below normal. The distinction between this condition and sclerema has already been indicated, and the **treatment** must depend upon a recognition of the pathological condition upon which the œdema is dependent.

Melæna Neonatorum (gastro-intestinal hæmorrhage) is a symptom of various morbid conditions of the gastro-intestinal mucous membrane. The most frequent of these is congestion due to asphyxia at birth, to pulmonary collapse, or heart-deformity. Asphyxia appears to be the condition most often determining melæna, and it has been shown, by experiments on animals, that it can produce extravasation into the gastric mucous membrane. Among other conditions the most common is ulceration of the œsophagus, stomach, or intestines. This has been attributed in some cases to venous stasis caused by asphyxia at birth followed by thrombosis; in others to destruction of follicles; in others to fatty degeneration of the arterioles. In others the cause has been sought in emboli derived from the ductus arteriosus, or from the umbilical vein. Extravasation beneath the gastric mucous membrane with subsequent rupture into the stomach has been observed, and in a few cases there seems to have been reason to attribute the bleeding to the hæmorrhagic idathesis. Septicæmic diseases must also be mentioned as occasional causes of melæna.

The **symptoms** are, as a rule, very pronounced. An infant, born to all appearance healthy, becomes without obvious cause blanched, collapsed, and somnolent. Sometimes the first symptom is vomiting of blood or blood-stained matter; in either case altered blood is soon passed from the intestine. The blood may be tarry, or clotted, and comparatively little altered in color. In other cases, when the bleeding is less rapid, the condition of anæmia and collapse is more slowly established. The bleeding may begin at any time within the first week or ten days of life, but most commonly on the second day. If copious, convulsions may occur, and the infant rapidly succumbs, death being preceded by extreme blanching of the surface, subnormal temperature, and stupor.

The **prognosis** in cases in which the symptoms become pronounced is grave. The mortality is probably about 60 per cent. But small hæmorrhages may occur into the intestinal canal, producing marked blackening of the fæces without serious consequences. Moreover, it should be remembered that the stools, or more often the vomited matter, may be stained with blood derived from the nose or naso-pharynx, or from cracks about the mother's nipple, which sometimes bleeds very easily.

If no local source for the blood can be found, the **treatment** should consist in the administration of cold liquid food (whey, predigested milk, iced broth); of the application of cold to the abdomen (ice-bag

or ice-cloths) and warmth to the extremities; and of the internal administration of stypties—gallic acid (gr. j every three hours), oil of turpentine (♏j in mucilage, every hour), ergotine (gr. ¼ to ½ every two hours). Extract of krameria (gr. ij every two or three hours) by the mouth, and injections of infusion of krameria (℥iv to v) into the bowel are recommended by Dr. Eustace Smith. Calcium chloride might probably be of service by increasing the coagulability of the blood.

Pemphigus Neonatorum occurs in two forms; syphilitic infantile pemphigus (*q. v.*) and a form which occurs under bad sanitary conditions, either sporadically in private houses, or endemically in lying-in institutions. The infant is well nourished, and the bullæ, which are numerous, appear a few days after birth on the pubes, thighs, buttocks, or around the mouth and chin—not on the hands or feet, which are the usual sites of syphilitic pemphigus. The infant should be removed to a healthy house, and if this be done will usually recover rapidly under the use of mild antiseptic applications.

[Several conditions in the new-born predispose to hæmorrhage, *e. g.*, delicacy of the blood-vessels, change in the circulation at birth. These hæmorrhages may be considered under two heads: (1) accidental or traumatic hæmorrhages, chiefly incidental to birth and (2) spontaneous hæmorrhages. The first group has been duly considered above, and also some of the second group, as separate diseases. But it would seem better to class these latter under one head, and to regard them with other spontaneous hæmorrhages, merely as symptoms of the infectious disease now generally known as *Hæmorrhagic Disease of the New-born*. This class of hæmorrhages occurs within the first few days of life; is spontaneous in origin, multiple in its location, occurring most often in the gastro-intestinal tract, navel, skin, and mouth, and is self-limited, running its course to death or recovery. While undoubtedly infectious in its nature, its exact cause is still unknown, bacteriological investigation not yet being conclusive.

It is of especial importance that the malady be distinguished from true hæmophilia, on account of the more favorable prognosis in the former; for we should not approach such cases with the idea that they may recover from this particular attack of bleeding only to die later from a slight cut. The main points in the differentiation of the Hæmorrhagic Disease of the New-born from hæmophilia are as follows: the rare occurrence of hæmophilia before the second year of life, with absence of a family history of bleeding, the occurrence of the Hæmorrhagic Disease nearly equally in both sexes, with elevated temperature, a self-limited course, and absence of recurrence.

Townsend gives a careful study of this disease in the Archives of Pediatrics, Vol. XI., No. 8.]

CHAPTER IV.

FOOD.

The Stomach and Intestines at Birth—Milk—Physiology of Digestion—The Quantity of Milk taken at Various Ages—Rate of Increase in Weight—[Management of Breast Feeding]—Artificial Feeding of Infants—Fresh Cow's Milk—Condensed Milk—Infant's Foods—The Bottle—Effects of Boiling—Bacteriology of Milk—Sterilization—Pasteurization—Milk Laboratories—[Modification of Cow's Milk—Indications for Modifying—Diet in Second Year].

The **stomach** of the infant at birth lies between the liver in front and the spleen, left adrenal, kidney, and pancreas behind. As the infant grows the fundus enlarges more rapidly than the rest of the organ, but, throughout early childhood at least, the normal position of the lesser curvature is vertical. At birth its capacity is about ℥j or ℥jss; at three months ℥iijss to ℥iv; after this age the rate of increase is slower, so that at the age of one year its capacity is about ℥ix. The rate of increase in the capacity of the stomach corresponds, therefore, fairly well with the rate of increase in the weight of the body.

At birth the length of the small **intestine** (9 ft. 5 in.) is about five times that of the large (1 ft. 10 in.) which is about the height of the body. During the first two months the growth of the small intestine is rapid, the increase being 4 feet.[1] At birth the sigmoid flexure forms nearly half the length of the large intestine, and one or more loops curve down into the pelvis. During the first three or four months the colon grows more rapidly than the sigmoid, which ceases to curve so far into the pelvis.[2] The cæcum in infants and young children often lies higher and more towards the middle line than in the adult.

Freshly drawn **milk** consists of a fluid part, the milk plasma, and of solid particles evenly disseminated through it. The solid particles are minute oil globules, probably coated with proteid, a varying number of small colorless cells without fat, and particles of casein and nuclein suspended in the fluid. Human milk is alkaline; that of cows, when quite fresh, either alkaline or amphoteric, though it is acid when it reaches the consumer. The quantity of milk secreted by a healthy woman varies very much, but, on the average, may be set down at a pint and a quarter to a pint and three-quarters in

[1] The figures are those given by Treves, *Brit. Med. Journ.*, 1885, i., p. 415.
[2] See Anatomical Constipation, p. 440.

twenty-four hours. The chief proteid constituents of milk are caseinogen and lactalbumen. The former is the precursor of casein, which is formed from it by the action of rennet, a ferment secreted freely by the stomachs of sucklings. At the same time some "whey-proteid" is formed and remains in solution. This curdling does not take place except in the presence of calcium salts (phosphate and

FIG. 2.

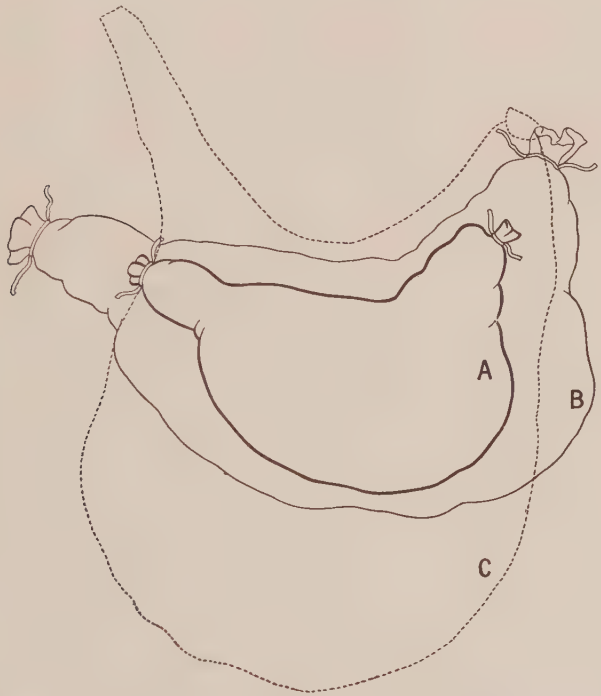


Diagram to illustrate the capacity of an infant's stomach. A, The smallest outline represents the stomach of an infant, age 5 days (capacity 25 c.c.; less than 1 fl. oz.). B, The intermediate outline represents the stomach of an infant, age 12 months (capacity 120 c.c.; about 4 fl. oz.). C, The dotted outline represents the dilated stomach of a rickety infant, age 7 months (capacity 300 c.c.; about 10 fl. oz.). (All $\times$ ¾.) After ROTCH (*Keating's Cyclopædia*).

chloride) which, however, are natural constituents of milk. In human milk the curd separates out in fine flocculi. Lactalbumen closely resembles serum albumen. It is slowly coagulated at temperatures between 70° and 80° C. (in cow's milk at 77° C. according to Halliburton). Milk is the product of the functional activity of the epithelium of the acini of the mammary gland, which in the active state of the gland are continually undergoing fatty change and disintegration, liberating the fat globules which float in the clear liquid secreted from the lymph. Lactose, or milk sugar, has only a

slightly sweet taste and is less soluble than sugar or dextrose. It readily undergoes the lactic-acid fermentation, and the lactic acid may subsequently be transformed into butyric acid. The chief salts of human milk are chlorides and phosphates of potassium, sodium, and calcium. The percentage composition of human milk shows slight individual variations, and is not constant in the same woman at all times, but the following may be taken to be a fair average :

Water.	Proteid.	Fat.	Sugar.	Ash.
87.7	1.82	3.94	6.23	0.31

The most important characteristics of human milk, and those by which it differs most from that of the cow are the low proportion of proteids and the high proportion of milk sugar. Franz Hofmann from the result of the recent series of analyses[1] made by him puts the percentage of proteid as low as 1.03.

Digestion of Milk.—The milk obtained by sucking is swallowed at once, and as the saliva and other secretions of the mouth are scanty, it reaches the stomach practically unaltered. The watery gastric secretion contains two ferments—rennet and pepsin—hydrochloric acid and mucus. Within a few minutes the rennet produces white flocculi of casein, in which much fat is entangled. The albumens of the milk are peptonized by the action of the pepsin and hydrochloric acid ; while, owing to the continuous secretion of the acid gastric juice, and perhaps to the conversion of a part of the milk-sugar into lactic acid, the acidity of the gastric contents increases progressively during digestion. When farinaceous foods are taken the increase is less rapid. The passage of the partially digested milk from the stomach into the duodenum begins early, and is completed in from one hour and a half to two hours after the suckling. When the food consists of cow's milk the time is longer. Intestinal digestion is estimated to last from six to eight hours. In the duodenum and the upper part of the jejunum admixture with the bile and pancreatic juice takes place, a bright yellow smooth pap being thus formed, and much of the fluid is absorbed. As the material passes along the intestine, digestion and absorption proceed, its bulk is reduced, and it becomes again more fluid, owing partly to solution of casein, and partly to the addition of the intestinal secretion. In the cæcum, by the gradual absorption of water and the addition of mucus, the orange-yellow, thick, pappy fæces, characteristic of normal digestion in the infant, are produced. The most important part of the intestinal digestion takes place in the small intestine, and especially in its upper part, although the absorption of fat still goes on in the colon. The fæces are acid, as indeed are the intestinal contents throughout.

[1] Made for Heubner (Penzoldt and Stintzing's "Handbuch," Bd. iv., s. 178).

In the healthy infant the digestion of milk is very complete. Chemical examination of the fæces shows that the whole of the sugar, nearly all, or more probably all the albumen, 96 to 98 per cent. of the fat, and nearly all the water (and presumably the salts) of the milk are absorbed. A considerable part, about a fourth, of the solid, dry contents of the fæces consists of fats—neutral fats, fatty acids, and soaps.[1]

The due performance of digestion depends upon the perfection of the two processes of secretion of the digestive fluids and the absorption of the products of digestion. Absorption is not merely a mechanical process due to osmosis and differences of pressure, but is brought about, in part, by the vital activity of the intestinal epithelium. The pancreatic secretion is essential to the due absorption of fat. It acts, apparently, by splitting up a part, probably a small part, of the neutral fats; the fatty acids thus produced form soda-soaps which facilitate emulsion, and the intestinal cells subsequently take up the fine emulsified particles. The absorption of fat by the epithelial cells takes place mainly in the upper part of the small intestine; it begins immediately after the entrance of the bile and pancreatic juice, and is continued throughout the jejunum. It is slow, occupying probably six to seven hours, but, in health, very complete, the fæces containing, as has been said, only from 2 to 4 per cent. of the fat taken.[2]

In a healthy infant the **motions**, passed usually thrice a day, are soft, homogeneous, of the consistency of cream or rather thicker, but not formed. Their color is bright orange or golden yellow. The odor is not of pronounced fæcal character, is, in fact, rather characteristic, and indescribable, never putrid. The passage of flatus with the stools is not the rule, but eructations, probably of air swallowed with the milk, are common after suckling. To this cause, also, attacks of hiccup, to which infants are very subject, may, with probability be attributed. They end sometimes in vomiting, which occurs very readily in infants. In many instances this rejection of milk must be accounted a physiological process, the stomach merely expelling some excess by which it has been over-distended. This expulsion of a part of the contents of the stomach is clearly unattended by nausea or pain, and the term *regurgitation* may, with advantage be applied to it. It is, indeed, a common saying that a "sick baby" is a healthy one.

The **quantity of milk** taken daily by a healthy infant at the breast

[1] O. Heubner in Penzoldt and Stintzing's "Handbuch d. Spec. Therap.," Bd. iv., s. 165.

[2] In Vaughan Harley's experiments on dogs (*Journ. of Phys.*, xviii., p. 1), the proportion absorbed eventually amounted to 85 per cent., but the animals had previously undergone a prolonged fast, which diminishes the power of forming the normal digestive secretions.

increases with its age. There are considerable variations in individual cases, but, adopting the estimate of Heubner,[1] we may take as averages :

Under 1 month	350	grammes,	or about	℥xj.
2 to 3 months	800	"	" "	℥xxviij.
Over 3 "	1,000	"	" "	℥xxxv.

A litre (1,000 g.) of human milk, according to the analyses of F. Hofmann, adopted by Heubner (Penzoldt and Stintzing's "Handbuch," Bd. iv., s. 178), contains

	Proteid.	Fat.	Milk-sugar.	Salts.
grammes	10.3	40.7	70.3	2.1
grains	159	628	1,085	32

or, in round numbers, two and a half ounces of milk-sugar, one and a half of fat, and one-third of an ounce of proteid.

During the first two or three days of life, before the secretion of milk is fully established, and for some days longer, the healthy infant is irregular in the frequency with which it suckles, but by the second week it becomes exceedingly regular. As a rule, it will suckle six, or at most seven times in the twenty-four hours, and at each suckling empties one breast. It will sleep seven to nine hours at night, so that roughly it suckles about every three hours during the day—*e. g.*, at 6 a.m., 9 a.m., noon, 3 p.m., 6 p.m., and 9 p.m. Some infants do well if suckled at both breasts at each meal, with a longer interval between the meals. But if an infant suckle at both breasts at each meal, with an interval of only two or three hours between the meals, it is an indication that the yield of milk is becoming inadequate either in quantity or quality. Before coming to this conclusion it is necessary to make sure that the infant is not suffering merely from thirst, either physiological or due to stomatitis, or from dyspepsia, which may lead it to desire to suckle frequently in the expectation of relieving the gastric discomfort which it experiences.

[In some instances, however, good results have apparently attended the adoption of this habit of giving both breasts every two or three hours. When the yield is insufficient the breasts from the more frequent stimulation are excited to greater activity and will gradually secrete a milk also richer in quality. It is possible in this way to convert a poor into a good breast-milk.]

The *length of time* for which it is customary to suckle an infant varies in different countries. In Germany, from nine to twelve months appears to be the rule ; in France and, probably, in England, the average is about twelve months or rather longer ; in Ireland, eighteen months or more ; and among negro races even longer.

[1] *Conf.* Feer (*Jahrb. f. Kinderhlde*, Bd. xlii., s. 195), who gives a series of interesting tables.

If the mother become pregnant, she should cease to suckle her infant. The occurrence of menstruation leads, as a rule, to a diminution in the quantity and to a deterioration in the quality of the milk; the infant loses weight and often suffers from diarrhœa, but this may be only a temporary disturbance. If menstruation have appeared early in the period of suckling, and if any loss of health which the infant may have shown is quickly regained it will usually be advisable to await a fresh appearance of the menses, which may be long delayed, or distinct evidences of pregnancy, before weaning. On the other hand, if the infant have been suckled already for nine or ten months it will usually be advisable to wean it. After nine months a healthy infant begins to desire other food, and has no difficulty in digesting well-cooked oatmeal and other cereal flours. At about one year it can take bread, fruit, meat, and fish in small quantities without disadvantage. In this, which is the natural mode of weaning, the infant is accustomed gradually to a mixed diet. A sudden change from an exclusive diet of breast-milk to a diet of cow's milk, broths, and cereals is undesirable, though many infants endure it without harm. When artificial food is first given it should be in a finely divided form, and attention should be directed to this precaution until the molars have been cut.

The symptoms of too prolonged lactation in the mother are weakness and disinclination to make ordinary exertions, sweating and anæmia, headache, backache, constipation. The child becomes anæmic and restless, and ceases to make weight.

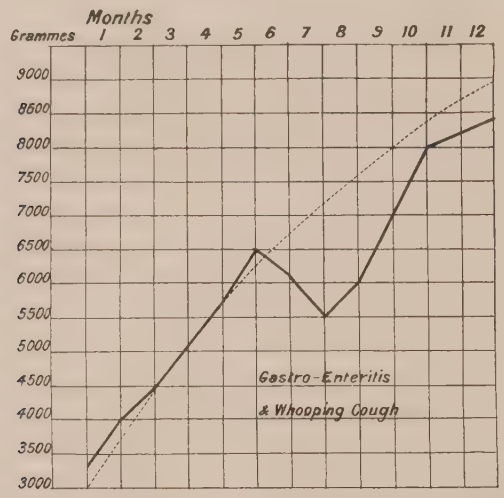
[Wherever there is unsatisfactory progress with an infant at the breast it should be an invariable rule to have made an analysis of the breast-milk as given in detail below.]

A healthy infant suckled by a healthy woman increases in weight with great regularity, but the rate of increase steadily diminishes (see page 18). A slight and short attack of illness produces a slight fall in the rate of increase, more serious or prolonged ill-health a more marked decrease or actual diminution, which may or may not be made good by an increased rate of increase after the reëstablishment of health. The curve on the next page, taken from one given by Sutils, shows the prolonged effect produced by an attack of gastro-enteritis complicated by whooping-cough. In infants fed artificially the rate of increase is less regular, and on the average it is smaller than in the infant at the breast, though, in those able to take and digest considerable quantities of carbohydrate foods, the contrary may be the case. Such children, however, are commonly unduly fat, and though they may weigh more they have less power of resistance to gastro-intestinal and febrile diseases than a breast-fed infant.

If the mother from any cause cease to be able to suckle her infant her place may be taken by a *wet-nurse*, but the practice is little fol-

lowed in Great Britain. The wet-nurse selected should be between twenty-three and thirty years of age, in the second or third month of lactation, of robust constitution, and free from any suspicion of tuber-

FIG. 4.



Curve showing the effect of gastro-enteritis complicated by whooping-cough on the weight. The dotted line shows the normal curve of increase. (After SUTILS.)

culosis, syphilis, or alcoholism. Her own child should be seen if possible, and she should not be accepted if it be in bad health.

[**Composition of Breast-Milk.**—Examination of many specimens of breast-milk has shown great variations in its composition, both in different women and in that of the same woman at different periods of lactation. During the first week the breast secretes a fluid somewhat different from that which is secreted later. At this period, it is called *colostrum*, from the presence of certain elements known as colostrum-corpuscles. These appear under the microscope as large cells; the fat globules are of larger size than they are later. Analysis of colostrum milk shows wide variations in its composition, indicating irregularity of mechanism in the mammary gland at this period. The breast has not yet got into regular working order; it is in a condition of unstable equilibrium.

The colostrum is thought to have a laxative effect and to aid in expelling the meconium. It normally disappears from the milk at the end of a week. Townsend has shown that its persistence is accompanied by failure to gain on the part of the nursling. It often reappears in the milk later in lactation when the breast is thrown into a state of unstable equilibrium from a variety of causes, *e. g.*,

pregnancy, return of menstruation or nervous excitement, and is invariably accompanied by digestive disturbance in the infant.

When lactation is well established, analysis of breast-milk shows on an average the following composition :

Reaction	Slightly alkaline.
Specific Gravity	1028–1034
Water	87–88 per cent.
Total Solids	13–12 per cent.
Fat	3–4
Sugar	6–7
Proteids	1–2
Total Ash	0.1–0.2

(Rotch.)

It must be remembered that these figures represent only *average* percentages of the elements of milk, and that we see breast-milk varying widely from the above and yet the infant fed thereon thriving and doing well ; an important point to which reference will be made later in discussing artificial feeding.

Nursing Habits.—The new-born infant should be put to the breast as soon after birth as possible, both that the breast may be stimulated to more immediate activity, and that the infant may at once use the instinct of sucking. Not much milk is secreted until the third day, but the infant will usually be satisfied with a five per cent. solution of milk-sugar, given in amounts of a half to one ounce every three or four hours for the first day or two of life. It is imperative that water be given freely in order to dilute the naturally concentrated urine and to flush out the kidneys. The administration of water at this period should be as much of a routine practice as the regular feeding established a little later. It should be given from a nursing bottle.

The young infant sleeps so much during the first few days of life that it will often be difficult to arouse him and to make him take the breast satisfactorily. Nevertheless the attempt should be made at least every three or four hours, and in some cases oftener. It is wise first to put him to the breast, then to give him his water ; in this way he will be more apt to nurse vigorously for ten or fifteen minutes, than if we have first satisfied him with water. Nursing once established, regularity should be rigidly enforced. We are all but bundles of habits, and it is as easy to establish good habits as bad in a healthy young infant. Irregularity in nursing has an injurious effect upon the child, both directly and indirectly ; directly by developing at the outset of his career irregular habits in the only direction in which he has habits, *e. g.*, eating and sleeping, and indirectly by the bad effect upon the breast-milk, too frequent nursings by repeated stimulation of the breast tending to produce a milk more concentrated than normal, and too infrequent nursings producing a

dilute milk. In the one case the nursling's digestive powers will be overtaxed, in the other his nutrition will inevitably suffer. He should be put to the breast every two hours during the first month or six weeks, then every two hours and a half till about the fifth month, after which, till the end of lactation, he may go three hours. We must remember, however, individual peculiarities in different infants, and expect to see many nurslings from the first go three hours and thrive upon this régime.

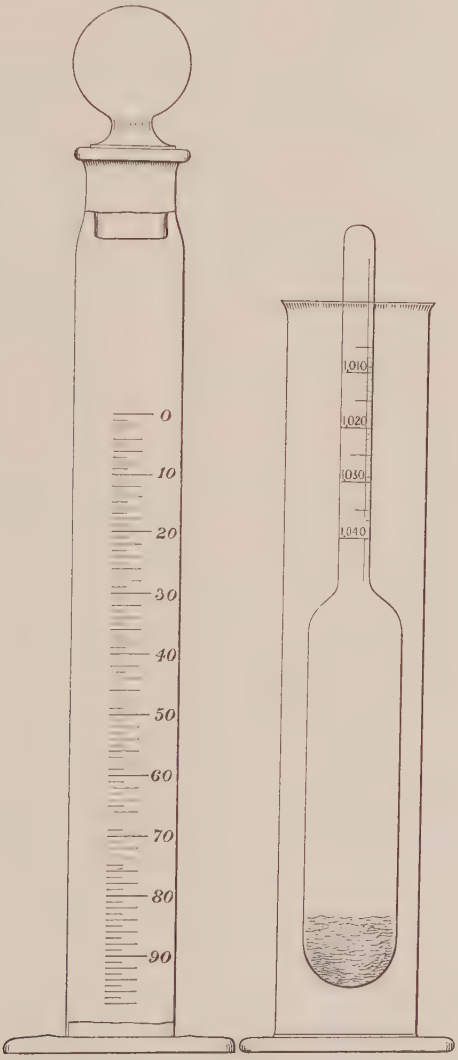
Regularity of habit is the essential point. The day feedings as a rule begin at 6 a. m., the last nursing being at 10 p. m. There should be but one night feeding, between 10 p. m. and 6 a. m. during the first two or three months, after which it may be omitted.

Control of Breast-Milk.—Many cases of breast feeding where the infant is thriving and gaining steadily in weight and all is going smoothly will require but little attention from the physician. But those cases, unfortunately numerous, which do not do well, will often give much trouble and require much attention to details by the physician himself. Too often the advice is given in an off-hand, careless manner to wean the baby and to give him some "baby-food," whereas before taking such an important step, especially in the early months of lactation, we should first investigate every case in all its phases, determine the trouble and its cause, and seek by all means in our power to correct the trouble and continue with the child at the breast. So much superior is breast feeding to all other methods that we should do our best to continue with it before resorting to substitute feeding.

The symptoms of difficult breast feeding on the part of the infant are generally fretfulness and dissatisfaction with the breast, prolonged nursing at each time, gastro-intestinal trouble manifested by vomiting, diarrhœa, or often by constipation and failure to gain in weight. The occurrence of any one of these symptoms should be a signal for thorough investigation of both mother and child. The general condition of the mother must be ascertained, also the details of her life, as to diet, exercise, habits of sleep, and general hygiene. Finally, but most important of all the breast-milk must be examined. While a complete chemical examination of the milk is desirable, it is not always possible to have such examination made. Holt, however, has devised a method of clinically examining breast-milk, a method of great practical value, of which I have made use in many cases with satisfaction. The principle of this method is based upon the fact that the specific gravity of breast-milk is modified solely by the amounts of proteids and fats, inasmuch as the salts are present in such small amount and the sugar is remarkably constant. Hence the percentage of fat and the specific gravity are obtained, and from these two data we make an approximate estimate of the proteids.

The method gives accurately the percentage of fat, but only approximately the proteids, showing whether they are about normal, much increased, or much diminished.

Fig. 3 B. Fig. 3 A.



The various steps are as follows. The milk for examination should be either the middle milk, obtained after the baby has nursed two or three minutes, or all the milk from one breast, the latter being more

desirable. Milk drawn at different times during a nursing varies in composition, the "fore-milk" being more dilute and the "strippings" more concentrated than the "middle" or average milk. The specific gravity is taken with any small hydrometer. (Fig. 3, A.) An ordinary urinometer will do, provided it be small enough. Fat lowers the specific gravity, other solids raise it.

The glass cylinder (Fig. 3, B), graduated in 100 parts and holding ten cubic centimetres, is then filled to the zero mark with the milk, allowed to stand eighteen or twenty-four hours, at the end of which time the percentage of cream is noted. The percentage of cream is to the percentage of fat as 5 : 3, *i. e.*, 5 per cent. of cream means the milk contains 3 per cent. of fat. If we wish to obtain the percentage of fat immediately, *e. g.*, in demonstrating at the clinic, Babcock's centrifugal machine may be used.

Having obtained thus the specific gravity and the percentage of fat, we estimate the proteids as follows:

		Calculated.
Normal fat.	Normal specific gravity.	Proteids normal.
High "	" " "	" increased.
Low "	" " "	" diminished.

From these data we infer that the proteids are about normal, much increased, or much diminished.

This method, of course, gives us information about the fat and proteids only; but it is these two elements which vary most in human milk, which give the most trouble in infantile digestion, and alone of the elements of milk in the present state of our knowledge can be modified by the diet and exercise of the nursing woman. The most usual variations from the normal are diminished fat, increased proteids, and diminished proteids. Rarely do we find the fats increased to such an extent as to cause the nursling trouble in digestion. The result of diminished fat is poor nutrition in the infant, with sooner or later the almost inevitable development of rachitis. Diminished proteids may lead to the same result. An excess of proteids overtaxes the infant's proteid digesting power and is apt to produce colic.

If the fat be found diminished, we may often increase it by giving the mother a diet rich in nitrogenous material. The same is true with regard to diminished proteids. An excess of proteids is generally due to an indoor, sedentary life and a generous table. Obviously the treatment for such a condition is exercise in the open air and regulation of the diet.

The urine of nursing women should be examined at least once a month, especially if it be found necessary to push the nitrogenous part of her diet to improve the breast-milk. An increase in nitrogenous food may result in renal irritation, if indeed it be not a factor in the production of actual organic disease in one predisposed thereto,

either by heredity or previous kidney trouble. The following case occurring in the practice of the reviser, shows the necessity of carefully watching the urine in nursing women: Mrs. A. B.—28 yrs.—primipara, had suffered for several years with headaches, her physician stating that they were not due to renal trouble. Pregnancy, delivery and convalescence superintended by the writer and normal in all respects, urine being examined repeatedly with negative results from the third month of pregnancy till two months after delivery. Baby, three months old, dissatisfied with breast-milk, examination of which showed fat 2½ per cent. More exercise in open air directed, amount of meats, eggs and milk increased, resulting in higher per cent. of fat in breast-milk. Baby seven months old, mother had severe headache similar to attacks several years before, from which she had been free during her pregnancy. Urine showed large amount of albumen, fatty renal epithelium, free fat, casts, hyaline, granular and fatty.

While it cannot of course be certain that the nephritis was caused alone by the richly nitrogenous diet of the mother, there can be no doubt that this diet was an important factor in its development. Had a monthly examination of the urine been made, the renal condition would have been discovered earlier and appropriate treatment instituted. The albumen gradually disappeared, but the urine (3 years later) contains a low per cent. of urea and the centrifugal sediment shows an occasional hyaline cast. No subsequent pregnancy. Child healthy and strong.

While the amount of sugar is usually constant at 6–7 per cent. occasionally it will be found below these averages and in such the nursling may fail to gain in weight. The following case illustrates this: A. B.—girl—weight at birth 7¾ lbs., gained steadily in weight up to fourth week, at which time she weighed 8¾ lbs. Analysis of mother's breast-milk was as follows:

Water	87.18
Total solids	12.11
Fat	4 per cent.
Proteids	2.78
Sugar	5.12
Ash	0.2

No change in régime advised. During the next four weeks she gained 18 oz., weighing at 8 weeks, 9 lbs. 14 oz. The breast-milk showed following analysis:

Total water	88.14
" solids	11.86
Fat	4.5 per cent.
Proteids	2.0
Sugar	5.1
Ash	0.2

No change in régime advised.

During the next three weeks baby gained but one ounce a week, only three ounces in all; thus it will be seen during first eight weeks, though gain was steady, it was small, averaging only about 4 ounces per week, and that in the next three weeks there was a decided falling off, practically no gain. As the fat and proteids were both high, this failure to gain more rapidly was probably due to the low per cent. of sugar, 5.1 per cent. And yet this deficit in sugar was not enough to give rise to apparent trouble. The weekly weighing alone showed something wrong.

She was given a solution of milk-sugar after each feeding, the amount needed being determined in the following manner.

Average capacity of stomach at 12 weeks . . .	96.6 c.c.
Amount milk taken (calculated)	96.6 "
	.07
Amount milk-sugar normally received calculated at the maximum 7 per cent.	6.762 g.
Amount milk-sugar actually received (5.1 per cent.) .	4.92 "
Deficit	1.84 g.
	about 30 grains.

She was, therefore, given 30 grains of milk-sugar in a drachm to a drachm and a half of water, after each feeding, a small amount of water being given to avoid distension of the stomach. This was the only change made in her daily routine of life. She immediately began to gain in weight, making 5 oz. in the next three days, and 2 oz. the next four days, 7 oz. for the week. The milk-sugar was continued for about 3 weeks, with average gain in weight of 7 oz. per week. It was then dropped, the amount of sugar in the breast-milk meanwhile having increased to 6.7 per cent. No explanation for this increase.

The infant was kept on the breast until she was nearly 11 months old, when she was weaned onto plain cow's milk, and at one year weighed 21 lbs.

The constipated habit in the mother, so unfortunately common in all women, is often the cause of disturbance in the nursling, even when analysis shows an average breast-milk. Excessive tea drinking by the mother is another common cause of trouble in the infant, manifested chiefly by peevishness, fretfulness, and a generally nervous state. Undoubtedly, under these two conditions, the mechanism of the breast is disturbed and it becomes an excreting, as well as a secreting organ, the system seeking to eliminate by this route toxic materials absorbed from the intestinal tract. Hence digestive disturbances arise in the infant, in many cases relieved by direct treatment of the mother, without any measures directed to the infant himself. Future investigations into the bacteriology of breast-milk

will, doubtless, give us information more exact than we at present possess in this direction.]

Artificial Feeding.—Stated broadly, the disadvantages of artificial feeding of infants are that, except under the most fortunate circumstances, the nutrition is less well maintained, and that there is a greatly increased liability to gastro-intestinal disease. The much higher mortality observed among hand-fed than among breast-fed infants is to be attributed to a combination of these causes, for it is obvious that an infant whose nutrition is imperfect, and who is already, perhaps, suffering from gastro-intestinal catarrh, will be much more liable to suffer from infective diarrhœa and to succumb to its effects.

Cow's milk, which is easily obtained at a moderate price, is the basis of most foods given to infants, though the milk of the ass presents certain advantages. The following table shows the percentage composition of human milk, cow's milk, ass's milk, and of certain mixtures, typical of many others, which may be used with most advantage to replace human milk.

	Proteids.	Fat.	Sugar.	Ash.	Water.
Human milk	1.82	3.94	6.23	0.31	—
Cow's milk	3.52	3.62	4.80	0.70	—
Cow's milk with an equal quantity of water	1.76	1.81	2.40	0.35	—
Cream mixture (Meigs)[1]	1.21	3.50	6.66	0.25	—
Fat milk (Gaertner)	1.76	3.00	2.40	0.35	—
Ass's milk	1.70	1.55	5.80	0.50	—

If Hofmann's estimate of the average amount of proteid in human milk (1.03 per cent.) be accepted, the excess in cow's milk is so great that even when diluted with an equal quantity of water there is still too much proteid.

The main differences in composition between human and cow's milk are that the latter contains more proteid and less milk-sugar. There are differences also in the salts, cow's milk containing more lime and less sodium chloride. The curd formed in the stomach from milk is denser the greater the proportion of casein and lime salts, and the higher the acidity. Cow's milk contains in round numbers twice as much casein, and six times as much lime; it is also usually acid. The curd formed from cow's milk is more bulky, less flocculent, and more disposed to form large clots than that formed from human milk. By diluting cow's milk with an equal quantity of water a fluid is obtained which contains about the right quantity

[1] [*Cream Mixture* (Meigs modified by Rotch).—Cream (about 15 per cent.) 2 parts; milk 1 part; lime water, diluted with three-fourth water, 2 parts; solution of milk-sugar (3 three-eighth drachms, water 3 fl. oz.) 3 parts. Or cream (20 per cent.) ℥jss; milk, ℥j; water, ℥v; milk-sugar solution as above, ℥iijss. Appendix.]

of proteid and yields a less dense curd, but it has too little fat, and only about half the proper quantity of milk-sugar. Many suggestions have been made for making good these deficiencies. The addition of barley water, which is very commonly practiced, adds a small quantity of carbohydrate, but its main object is to cause the curd to be more flocculent. In Meigs' *cream mixture*, and the various modifications of it, the defect in the quantity of fat is made good by the addition of cream, that in the quantity of milk-sugar by the addition of a solution of that substance. Gaertner's "fat-milk" is made by dividing into two equal parts, by means of the separator machine, a bulk of milk diluted with an equal quantity of water; one-half of the yield contains nearly all the fat and half the proportion of proteids, milk-sugar, and salts contained in the original cow's milk. This milk contains too little milk-sugar, and to make good this Cautley has suggested that the milk should be diluted with a solution of milk-sugar instead of with water, before separation. Sexhlet, in order to avoid certain difficulties in sterilization, has suggested the addition of a quantity of milk-sugar sufficient to compensate not only for the deficiency in milk-sugar but also for the deficiency in fat; the composition is based on the fact that 243 parts of milk-sugar are required to yield the same amount of work as 100 parts of fat. This suggestion, however, ignores the physiological differences between a carbohydrate and a hydrocarbon, and its utility is doubtful, at least for a permanant diet.

Condensed Milk is used very extensively for the artificial feeding of infants. Its main advantages are that it is cheap and handy, and that if prepared fresh for each feeding with boiled water the fluid which the child takes is, if ordinary care be exercised, free from decomposition and practically almost sterile. The main disadvantages attending its use are that it is a cooked food, and that, therefore, a too exclusive reliance upon it will tend to produce a scorbutic condition, and that, even with the best brands, the quantity of fat in the dilution ordinarily used is too low. Very many brands of condensed milk sold are made from separated milk and certain very small proportions of fat (cream); many also are loaded with cane-sugar intended to prevent decomposition, to cover any disagreeable taste, and to increase the bulk. Instructions should, therefore, be given that the brand of condensed milk selected should be guaranteed, by a statement on the cover or tin, to contain the whole of the cream of the original milk, and to be free from added sugar. The degree of dilution to be recommended must be governed by a consideration of all the circumstances, and will always be the result of compromise. The most careful dilution of the best condensed milk will yield a fluid which will contain too much proteid matter and too little fat. When condensed milk disagrees, an attempt may be made to over-

come the difficulty by increasing the dilution and adding milk-sugar. The use of condensed milk in temperate climates should, however, always be looked upon as no more than a convenient temporary expedient to bridge over an interval of time during which the milk supply is under suspicion. In hot climates, and in India especially, where the native indifference to cleanliness constitutes a peculiar difficulty, condensed milk may be much preferable to any other obtainable supply. For such climates, dry, powdered milk appears to be particularly suitable.[1]

A great number of *patent foods* are offered for sale, for most of which it is claimed that they are "perfect substitutes" for mother's milk. The claim can in no case be substantiated, and in many instances is in glaring disagreement with the chemical constitution of the preparation. Many, for instance, contain large quantities of unconverted starch, and should, therefore, be absolutely rejected. In others, either the whole of the starch has been converted into dextrose, maltose, etc., or it has been in part converted, and the preparation contains sufficient diastase to ensure complete conversion during the process of cooking. Such preparations when well made, as many of them are, are useful adjuncts to cow's milk. They supply the deficient carbohydrate in a form usually found more palatable than milk-sugar, and being easily prepared they are not likely to be allowed to undergo decomposition owing to being kept after mixing.

The kind of bottle used is a point of considerable importance, and one to which sufficient attention is often not given. The modern bottle with a long tube well deserves the name often applied to it in France, *tue-bébé*—the baby killer—and its use is forbidden by law in some countries. The main reason for the favor in which it is held is that a full bottle can be prepared and the infant left in its cradle to suck at its own will. An infant in good health will not swallow at one time more than is good for it, but a slight attack of dyspepsia or stomatitis, causing sensations of discomfort in the stomach or mouth, will induce it to go on sucking until the bottle is empty, and thereafter to continue sucking and swallowing air, with the result that it begins to suffer from vomiting and flatulent colic. These are, however, minor objections. The great evil of the long-tubed bottle is the practical impossibility of keeping the tubing clean and free from particles of decomposing curd. Every meal the infant gets is thus inoculated with the causes of decomposition, and the liability to gastro-intestinal disorder is greatly increased; the old boat-shaped bottle is much to be preferred. It is itself of a form which renders it much more easily cleaned, it has no long tubing, and as it must be held in the nurse's hand while the infant sucks, the meals are more likely to be taken at regular intervals, and to be of a proper quantity.

[1] A dried milk is prepared by Messrs. Allen and Hanburys.

The **effect of boiling** on milk is to precipitate the lactalbumen, which rises as a scum to the surface, taking with it some of the fat and caseinogen; to alter the caseinogen so as to render it less readily curdled into casein; to cause the fat globules to run together into larger drops; to alter its flavor; and to give it a darker (brownish) color. None of these changes occurs rapidly, and milk which is merely brought to the boil, though it is distinctly altered in taste, loses little of its lactalbumen, does not change in color, and the perfection of the emulsion of the fat is little diminished. On the other hand, the casein clot formed by rennet is more flocculent than that obtained from fresh milk, owing to a part of the dissolved calcium salt being precipitated as tri-calcium phosphate.

The milk secreted by the mammary gland in health contains no microbes or a very small number. At most the first few drops contain a considerable number washed, probably from the orifices of the ducts. Cow's milk, however, becomes quickly contaminated by impurities on the teats of the animal, the hands of the milker, and the vessels into which it is received, by atmospheric dust, and in various ways during the manipulations through which it passes before reaching the consumer, among which must be included the addition of water.

The diseases of which cow's milk may be the vehicle are (1) *Tuberculosis:* the milk of a cow suffering from tuberculous disease of the udder contains the bacillus tuberculosis in a very virulent state. (2) *Typhoid fever*, the virus of which may find access to the milk in various ways, but mainly, probably, through added water. (3) *Diphtheria.* (4) *Scarlet fever.* (5) Certain forms of *diarrhœa*, including acute summer diarrhœa.

Milk which has been carelessly handled by the dealers contains an enormous number of **microbes.** By the time it reaches the consumer there may be one or two or three millions in a drachm, and even this last number has been exceeded. In much of that supplied in towns, especially by the smaller retailers, lactic-acid fermentation has already commenced, while among the microbes which it contains are some capable of breaking up the proteids of milk with the production of poisonous bodies—alkaloids and peptones. It is to the irritating qualities thus imparted to milk that diarrhœa produced immediately by its ingestion must be attributed, while the general toxæmia by which death is brought about in the more acute cases is due to the absorption of the poisonous products of proteid decomposition.

The bactericidal powers of the gastric juice secreted by a healthy stomach[1] afford some protection against the continuance within the gastro-intestinal canal of fermentations and decompositions which have

[1] Soltau Fenwick states ("Disorders of Digestion in Infancy and Childhood," London, 1897) that in the infant's stomach free hydrochloric acid can be detected only at the end of digestion, and that the development of bacteria may therefore go on in its stomach unchecked by the gastric juice.

commenced in the milk before ingestion, but the toxic bodies which such milk contains are absorbed, while its irritating qualities produce catarrh, and such an alteration in the secretions as diminishes or destroys their power of checking microbial growth. In consequence, fermentations and decompositions which have commenced in the milk before ingestion continue within the gastro-intestinal system.

In the healthy infant on a milk diet Escherich found that two microbes predominated in the fæces, the b. lactis aerogenes and the b. coli communis. They produce fermentation of the milk-sugar with the production of lactic and acetic acid, and gases (CO_2, and H). In diarrhœa a very large number and variety of microbes are present in the fæces. Booker distinguished no fewer than 33, many or all of which were capable of causing decomposition with the production of toxic bodies. Thus, severe acute diarrhœa may be produced by saprophytic microbes, though it is probable that certain forms of acute summer diarrhœa are due to infection of the intestines by specific microbes. Such microbes have been described by Lesage in acute green diarrhœa, and by Flügge, who believes that he has identified, in a spore-bearing bacillus, the cause of acute summer diarrhœa in one of its forms. This bacillus is spore-bearing, and its spores are not destroyed by exposure to a temperature of 100° C.

By raising milk to the boiling point of water all the bacteria which it contains are destroyed with the exception of the resistant spores of certain of them. Absolute sterilization can be obtained only by prolonged heating at 100° C., or by fractional sterilization. For ordinary purposes, when the milk need not be kept for more than twenty-four hours, this complete sterilization is not necessary, and the term **sterilized milk** may be applied conveniently to milk which has been freed by heat from the adult forms of bacteria. A large number of apparatus have been devised for this purpose. There are two main types: (1) Those in which the milk is sterilized in bulk, and (2) those in which the quantity for each feeding is sterilized in a separate bottle. Woodhead gives the following indications for sterilizing milk for domestic purposes. The vessel in which the milk is contained should be placed in a saucepan containing a quantity of cold water equal to the bulk of the milk to be sterilized. The vessel should be provided with a stirrer to be used from time to time so as to maintain an even temperature and the diffusion of the cream. "The water should be boiled over a good brisk flame in order that the best results may be obtained, and the heating process should be continued until the temperature throughout the milk has risen to from 88° to 92° C.; in most cases this takes place at the end of about twenty-five minutes; but in order to be perfectly safe it may be recommended that every quart of milk

treated in this fashion should be heated for half an hour; that is, for about twenty minutes after the water in the outer pan has begun to boil." These conditions are fulfilled in a sterilizer designed by Cathcart of Edinburgh, in which the day's supply can be sterilized at once for one child. As will be seen from Fig. 5, it is provided with a draw-off tap and with a stirrer, by means of which the cream can be diffused through the milk on each occasion before the milk is drawn off. Hunter Stewart,[1] working with this apparatus, found that after half an hour on the fire the temperature of the milk was 91° C. (196° F.), and that samples drawn off by the stopcock at

Fig. 5.

Cathcart's Sterilizer. A cylindrical block-tin vessel tapering slightly towards the base, and provided with a tap at the bottom, through which the milk is drawn off. The lid fits tightly, and the line of junction with the can is rendered air-tight by an elastic band, which is slipped over after sterilization is completed. The lid has a wide, funnel-shaped aperture through which the milk can be introduced; the aperture is then closed by a plug of cotton-wool. The sterilizing can is placed in a saucepan, of capacity sufficient to receive it conveniently, and containing cold water. This is placed on a brisk fire for half an hour. A special feature of the invention is the stirrer, a screw-shaped piece of tin provided with a long handle which projects through the aperture in the lid; by its use the milk can be stirred from time to time during the process of sterilization, to ensure a uniform temperature throughout the milk.

two, four, twenty-four, and forty-eight hours after treatment were all sterile. The special advantage of this apparatus is that risks of contamination of the milk after sterilization are reduced to a minimum. Aymard's sterilizer, in which also the milk is sterilized in bulk, consists of an outer steam-chamber and an inner receptacle for the milk provided with a separate lid, and a spout which passes through the steam-chamber, to open on the outside. When ready for use the covered milk-chamber is enclosed entirely within the outer steam-chamber. In the smaller sizes for domestic use the steam is generated by heating the water at the bottom of the

[1] *Brit. Med. Journ.*, 1896, vol. ii., p. 626.

outer chamber to the boiling point over a gas or other stove. The milk reaches a temperature of 200° F. in about ten minutes after the water has begun to boil. The advantages of the apparatus are that it is simple and easily cleaned, that there is little separation of cream or formation of scum, and that owing to the milk vessel being entirely surrounded by steam, there is little alteration in the taste or smell of the milk.

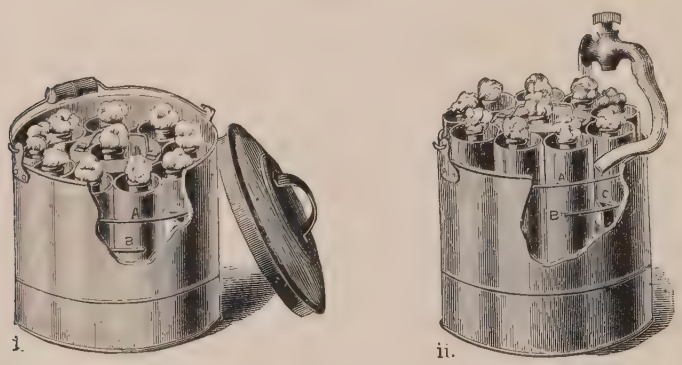
Soxhlet's apparatus may be taken as the type of those in which the quantity of milk required for each feeding is sterilized separately. The advantages of this method are that when properly carried out the contents of each bottle when given to the child have not suffered any contamination after sterilization, and that the milk may be diluted or otherwise modified in various ways before sterilization. The apparatus consists of a covered saucepan into the bottom of which a sufficient quantity of cold water is introduced. The bottles are placed in a wire-work carrier. Each bottle is closed by an india rubber disc, held in place by a metal cap. The carrier is supported in the saucepan a little above the level of the water. The saucepan is then heated. Thus, when the water in it begins to boil, the bottles are surrounded by steam. The can is kept on the stove about three-quarters of an hour. It is then removed and the bottles cooled rapidly by running cold water into the can. The loose india rubber disc permits the escape of steam from the bottles during sterilization, but during the rapid cooling the india rubber comes into contact with the rim of the bottle, and by the fall of pressure within the bottle, becomes tightly applied. If, then, the bottle be properly sealed the upper surface of the india rubber disc is concave. When required, the bottle is warmed, unopened, by placing it in hot water. The india rubber disc is now removed, and as this is done a hissing noise of air entering the bottle ought to be heard. The teat, which should be kept in boric-acid solution (5-per-cent.) when not in use, is slipped over the neck of the bottle, which is then ready for use. Any milk remaining in the bottle after the meal should not be used again.

The main objections to the use of sterilized milk, over and above the possibility that its continuous use may cause scurvy (*q. v.*) are: (1) That its taste and smell are altered—an objection which is minimized in the two apparatus for sterilization in bulk described above, and the force of which is further lessened by the fact that infants soon become accustomed to it; (2) that the greater part of the carbonic acid gas is driven off, which involves an alteration in the composition of the phosphates, a precipitation of calcium and magnesium carbonates, and a diminution of the ease of digestion; (3) that the emulsion of the fat is less perfect than in raw milk, and that the fat globules tend to coalesce into drops; (4) it has been asserted

also that the casein is less rapidly digested, though this statement has been contradicted. The third objection applies with special force to cream mixtures.

To obviate some of these objections, a process to which the term **pasteurization** is applied has been devised. The main advantages claimed for it are that the condition of the fat is not altered, that the taste and smell are not changed permanently, that the digestibility of the casein is little diminished, and that less carbonic acid is driven off. Milk kept at a temperature of 65° C. (149° F.) for thirty minutes is freed from all microbes which can be cultivated by the plate. These include the bacillus diphtheriæ (Klebs-Loeffler), Eberth's typhoid bacillus, and the cholera vibrio. The only doubt is with regard to the bacillus of tuberculosis, which, however, does not long resist a temperature of 70° C. (158° F.). Spores will also, of course, escape. On the whole, it may be said that pasteuri-

Fig. 6.



i. ii.

Freeman's Pasteurizer.—i, apparatus arranged for heating the milk before the pail is covered; ii, apparatus arranged for cooling the milk; A, wire binding the cylinders together, which rest on the support C when the milk is being heated; B, one of three short wires which rest on C when the receptacle is raised for cooling, as shown in ii. To use: Fill the pail to the groove with water; boil; remove from stove; put in milk receptacle (each cylinder must be filled with cold water, whether it contains a bottle of milk or not); cover, and leave standing on non-conductor for three-quarters of an hour. Open, raise receptacle, and cool from faucet.

zation at 70° C. will render milk so far sterile that it will remain free from decomposition for at least twenty-four hours, which is as long as it is, under ordinary circumstances, desirable to keep milk.

The process may be carried out[1] in a tin vessel, with a tightly-fitting lid through which a thermometer passes. The bottle, or series of small bottles, containing the milk should stand upon a wire grating. Cold water is introduced into the vessel, and the temperature raised slowly to 160° F. (71° C.). The vessel is then taken off the fire and kept under a thick cosy for thirty minutes. At the end of this time the bottles should be taken out and cooled rapidly. Freeman,

[1] Johnstone Campbell, *Brit. Med. Journ.*, 1896, vol. ii., p. 623.

of New York, has introduced an ingenious apparatus for domestic use. In it a known quantity of water is raised to the boiling point in a suitable covered pail. The bottles of milk, which are stoppered with cotton-wool, are placed in a carrier consisting of a series of metal cylinders, and introduced into the pail, which has been removed from the fire. The milk is kept in the covered pail for three-quarters of an hour. During the first quarter of an hour the temperature of the milk in the bottles rises to 65°–68° C., and a degree or two higher in the next ten minutes, and then falls very slowly, so that when the can is opened at the end of forty-five minutes it is still above 65° C. The bottles are cooled rapidly by the introduction of cold water into the pail, and are kept in a refrigerator until required to be warmed for use.

The processes of sterilization and pasteurization are, however, designed to remove microbial contamination, which it would obviously be very much better to prevent. Experience shows that, with proper precautions, milk can be collected which, without any preparation, will keep sweet and palatable for a much longer period than usually intervenes between the hour of milking and the consumption of the milk. In the *milk laboratories* which have been established in various cities of America this has been done, and at the same laboratories the milk is so modified under skilled supervision that it contains precisely the proportions of fat, proteid, and carbohydrate which the physician may prescribe. Such modified milk, when obtained under proper guarantees as to the health of the milkers and the freedom of the herd from tuberculosis, and as to constancy of the composition of the samples supplied, appears to be a substitute for human milk as nearly perfect as can be devised.

[**Milk Laboratories.**—The work at the milk laboratories consists essentially in the preparation of an artificial human milk, and, as already said, the adaptation to the infant's digestive power of requisite amounts of fat, proteids, and sugar. The basis of such preparation is cow's milk. The problem is the conversion of cow's into human milk. For the elaboration of the methods by which this is accomplished, and by which substitute infant feeding is put upon a scientific basis, the world is indebted to Rotch.

A comparison of average analysis of cow's and woman's milk shows several points of difference, as evidenced by the following tables.

	Woman.	Cow.	
Reaction .	Slightly alkaline.	Slightly acid.	
Water . .	87–88 per cent.	86–87 per cent.	General averages.
Total Solids .	13–12	14–13	
Fat . .	4.00	4.00	
Milk-sugar .	7.00	4.50	
Proteids .	1.50	4.00	
Ash . .	0.2	0.7	

Briefly the problem is to convert a preparation slightly acid and comparatively concentrated into one slightly alkaline and more dilute. More in detail we must modify the cow's milk as follows: the reaction must be made slightly alkaline, the total water increased, the total solids diminished, the fat be kept the same, the sugar increased, proteids diminished. The various steps by which this modification is made are as follows: separation of fresh cow's milk into cream and milk, recombining these in various proportions, adding water, milk-sugar, and lime-water. The more accurately we can take these various steps, the better will be our results, and we have in the milk laboratory an institution where this modification is done quickly, accurately, and scientifically.

A case best illustrates the principle upon which we proceed. An infant six months old, deprived of his mother's milk, is to be put on modified milk. We write a prescription as follows:

℞

	Per Cent.	Remarks.	
Fat	4 00	Number of Feedings	6
Milk-sugar	7 00	Amount at each Feeding	℥vj
Albuminoids	1 50	Infant's Age	6 mos.
Mineral Matter	—	Infant's Weight	16 lbs.
Total Solids	12	Alkalinity	6 per cent.
Water	88	Heat at	155°F.
	100 00		

Ordered for Baby X.....

Date, Signature,

.....January 1st,.....189 . .....A. B. Smith, M. D.....

This prescription is sent to the laboratory and there from formulæ carefully worked out, definite amounts of milk, cream, water, milk-sugar and lime-water are combined to produce the required percentages. The resulting mixture is then divided among six tubes or bottles, corresponding to the number of feedings ordered; these are stoppered with aseptic cotton, placed in a basket, heated for ten or fifteen minutes at the required temperature, 155° to 160° F., and then shipped to the baby ready for use. Each bottle should be heated to the body-temperature immediately before feeding.

It will readily be seen that by varying the amounts of the different ingredients used, we can produce many combinations of percentages. We thus have great latitude in prescribing and can vary our prescription to suit the needs of the individual case.

As has been already stated, the proportions given in the above analysis of breast-milk represent a general average and we see infants fed on a breast-milk differing widely from this average, yet healthy and thriving. We should take the hint and be prepared to vary our prescription in different infants.

The number and amounts of feedings will vary with the age of the infant, and, as in breast-feeding, we cannot lay down any hard and fast rules which will fit all cases. The intervals between the feedings should be the same as already given for breast-feeding. The amount at each feeding may be stated broadly as follows:

Age.	Amount.
1–2 weeks	1–1½ oz.
2–4 "	2–3 "
2–3 months	3–4 "
4–6 "	4–6 "
6–8 "	6–7 "
8–10 "	7–8 "
10–12 "	8–10 "

Each infant, however, must be a law unto himself, and some will require more, others less, than the above amounts at the respective ages.

Home Modification.—While the modification of cow's milk is done most accurately at the laboratory, this institution is at present established only in some of our large cities, and hence the modification must oftener be done at home. This can be accomplished with much accuracy, either by the use of the following tables, for which I am indebted to Rotch, or by the use of an apparatus known as the "Materna," devised by Dr. S. V. Haas, and now for sale at the large chemical supply companies of our cities. In home modification the cream should be a 20% cream, obtained from any good dairy, and the milk-sugar should first be dissolved in the water. The total amount can, of course, be doubled or tripled, according to circumstances.

Tables (Rotch).

Table 1.

Fat	1.00	Cream	2 ounces.
Sugar	5.00	Milk	2 ounces.
Proteids	0.75	Lime-water	1 ounce.
Lime-water	5.00	Water	15 ounces.
			20 ounces.
		Milk-sugar	2 measures.[1]

[1] Each measure contains 3⅜ drachms.

Table 2.

Fat	2.00	Cream	4 ounces.
Sugar	5.00	Milk	None.
Proteids	0.75	Lime-water	1 ounce.
Lime-water	5.00	Water	15 ounces.
			20 ounces.
		Milk-sugar	2 measures.

Table 3.

Fat	2.00	Cream	4 ounces.
Sugar	5.50	Milk	1½ ounces.
Proteids	1.00	Lime-water	1 ounce.
Lime-water	5.00	Water	13½ ounces.
			20 ounces.
		Milk-sugar	2¼ measures.

Table 4.

Fat	2.50	Cream	5 ounces.
Sugar	6.00	Milk	None.
Proteids	1.00	Lime-water	1 ounce.
Lime-water	5.00	Water	14 ounces.
			20 ounces.
		Milk-sugar	2½ measures.

Table 5.

Fat	3.50	Cream	7 ounces.
Sugar	6.50	Milk	1 ounce.
Proteids	1.50	Lime-water	1 ounce.
Lime-water	5.00	Water	11 ounces.
			20 ounces.
		Milk-sugar	2½ measures.

Table 6.

Fat	4.00	Cream	8 ounces.
Sugar	7.00	Milk	None.
Proteids	1.50	Lime-water	1 ounce.
Lime-water	5.00	Water	11 ounces.
			20 ounces.
		Milk-sugar	2¾ measures.

Table 7.

Fat	4.00	Cream	8 ounces.
Sugar	7.00	Milk	2½ ounces.
Proteids	2.00	Lime-water	1 ounce.
Lime-water	5.00	Water	8½ ounces.
			20 ounces.
		Milk-sugar	2½ measures.

Table 8.

Fat	4.00	Cream	8 ounces.
Sugar	7.00	Milk	5 ounces.
Proteids	2.50	Lime-water	1 ounce.
Lime-water	5.00	Water	6 ounces.
			20 ounces.
		Milk-sugar	2½ measures.

Table 9.

Fat	4.00	Cream	8 ounces.
Sugar	7.00	Milk	7½ ounces.
Proteids	3.00	Lime-water	1 ounce.
Lime-water	5.00	Water	3½ ounces.
			20 ounces.
		Milk-sugar	2 measures.

Table 10.

(For weaning.)

Fat	4.00	Cream	8 ounces.
Sugar	5.00	Milk	7½ ounces.
Proteids	3.00	Lime-water	1 ounce.
Lime-water	5.00	Water	3½ ounces.
			20 ounces.
		Milk-sugar	1 measure.

Table 11.

(For weaning.)

Fat	4.00	Cream	8 ounces.
Sugar	5.00	Milk	8 ounces.
Proteids	3.25	Lime-water	1 ounce.
Lime-water	5.00	Water	3 ounces.
			20 ounces.
		Milk-sugar	⅞ measure.

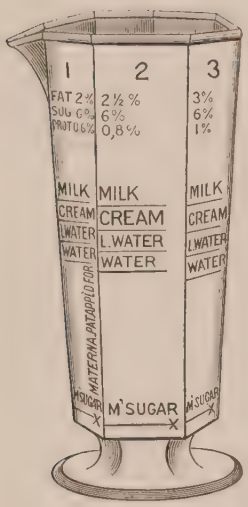
Table 12.

(For weaning.)

Fat	4.00	Cream	8 ounces.
Sugar	4.50	Milk	12 ounces.
Proteids	3.50		
			20 ounces.

The milk thus prepared is now distributed among the requisite number of bottles, the bottles plugged with absorbent cotton and pasteurized as described on page 72.

FIG. 7.



Haas' "Materna" (see cut) is an ingenious apparatus and simplifies further the method of home modification. While it does not of course allow the range of prescribing found in Rotch's tables, and so cannot be used for the more gradual changes necessary in ill health, when the formulæ coincide it simplifies home work. The apparatus consists of a glass vessel, and is made in three sizes, containing respectively 16, 20, and 24 ounces. The external surface is divided by vertical lines into seven sides or "panels," on each one of which is indicated by horizontal lines the amount of milk-sugar, water, lime-water, cream, and milk to be used to produce the percentage of fat, proteids and sugar marked on the same panel at the top. The table shows the arrangement of these panels.

1	2	3	4	5	6
Fat, 2% Proteids, 0.6% Sugar, 6%	Fat, 2½% Proteids, 0.8% Sugar, 6%	Fat, 3% Proteids, 1% Sugar, 6%	Fat, 3½% Proteids, 1½% Sugar, 7%	Fat, 4% Proteids, 2% Sugar, 7%	Fat, 3½% Proteids, 2½% Sugar, 3½%
Milk	Milk	Milk	Milk	Milk	Milk
Cream	Cream	Cream	Cream	Cream	Cream
Limewater	Limewater	Limewater	Limewater	Limewater	Barley gruel
Water	Water	Water	Water	Water	
Milk sugar	Milk sugar	Milk sugar	Milk sugar	Milk sugar	Gr. sugar
X	X	X	X	X	

Six different combinations can thus be made, and even more as suggested by Haas, by combining 16 ounces of one panel with 16 ounces of another. The total amount for the day having been prepared is then distributed in bottles and pasteurized as already described.

Indications for Modifying.—The necessity of wide variations in modified milk has already been mentioned. Particularly in infants acutely sick and in those not thriving from more or less chronic digestive disturbance, must we be prepared to vary our modification according to the indications arising in each case. As to exactly what those indications are, we have yet much to learn. Confronted with a case of difficult feeding, the question we ask ourselves is: is this child suffering from proteid-indigestion, from fat-indigestion, or from sugar-indigestion? For it is one or more of these three substances which is invariably the cause of the trouble. We often hear it said that "the milk" does not agree with the baby. That is as irrational and as indefinite as to say that "food" does not agree with an adult. Which part of the milk, of the food, is at fault? The mistake arises from regarding milk as a simple food, whereas in reality it is a very complex food, consisting as it does of fat, sugar, proteids, salts and water, essentially the same elements it will be noted which make up the diet of the adult, and, as in a case of "indigestion" in the adult, we ask whether it is the carbohydrates or the nitrogenous part of the food which is causing the trouble, so in the infant we seek to find out whether he be suffering from fat-indigestion, sugar-indigestion, or proteid-indigestion.

An excess of fat is apt to cause vomiting soon after feeding; occasionally, also, bits of fat are found in the dejections. It is rare, however, to find trouble arising from a comparatively high percentage of fat. Infants as well as children demand and assimilate large amounts of this element. Too little fat is shown by dry constipated stools, slow gain in weight, and, if long continued, the development of rickets.

An excess of proteids is indicated by colic and the presence of curd in the dejections.

An excess of sugar is apt to be accompanied with eructations of gas and also with considerable flatulence. Holt believes also that an excess of this element causes colic and green, acid, thin stools. We also see soft, flabby flesh in infants taking much sugar; too little of the element results in slow gain in weight.

More clinical data and a more thorough knowledge of physiological chemistry, however, are needed before we can lay down exact rules for prescribing these three important elements of food.

The following cases illustrate some of the points mentioned above.

The first case was an infant, female, weight at birth 6 lbs., fed on barley and milk, Horlick's & Carnrick's food, up to two months of

age. The condition then one of marked atrophy, but without marked digestive disturbance. Her subsequent history is shown in the following table:

Date.	Age.	Feeding.			Weight.
		Fat.	Sugar.	Proteids.	
April 21.	2 months.	2.50 %	6.00 %	0.55 %	6 lbs. 6 oz.
" 24.	———	"	"	"	7 " 12 "
" 27.	———	2.75	6.00	0.75	8 " 2 "
" 30.	———	"	"	"	8 " 6 "
May 5.	———	"	"	"	8 " 10 "
" 13.	———	3.00	7.00	1.00	9 " — "
" 20.	3 months.	3.25	7.00	1.00	9 " 8 "
" 25.	Rötheln & Colic.	3.25	7.00	0.75	
June 27.	4 months.	3.25	7.00	1.00	
July 16.	———	3.50	7.00	1.00	12 lbs.
" 31.	5 months.	4.00	7.00	1.00	12 " 8 oz.
Aug. 28.	6 months.	"	"	"	14 "
Sept. 9.	———	"	"	1.25	
" 16.	First	Tooth.	———	———	———
" 21.	7 months.	4.00	7.00	1.50	
Oct. 6.	———	4.00	7.00	2.00	
" 12.	———	4.00	7.00	2.25	
" 26.	8 months.	"	"	"	14 lbs. 12 oz.
Nov. 11.	———	"	"	2.75	
" 27.	9 months.	"	"	3.00	
Dec. 31.	10 months.	"	6.00	3.50	
Jan. 6.	———	"	"	4.00	
" 13.	11 months.	Plain	cow's milk.		19 lbs.

This case did uninterruptedly well. The slow gain during September and October was undoubtedly due to the low proteids, and probably that element could have been pushed more rapidly in this particular case, though as a rule we must be cautious in increasing this constituent.

The child is now at four years strong and hearty.

The next case was more difficult and made less steady progress.

A male infant, age two months, with a nervous, hysterical mother, whose milk showed low fat and high proteids (Holt's method). Infant suffering constantly from intestinal colic and gaining only slowly in weight. After a vain attempt to improve the quality of the mother's milk, I stopped it at once, and, mindful of the baby's weak proteid-digesting power, put him on: fat 2 per cent., sugar 6 per cent., proteids $\frac{1}{4}$ per cent. He stopped crying at once, gained slowly in weight, the fat being increased every third day, the proteids kept at $\frac{1}{4}$ per cent. until the tenth day, when he was given and took well: fat $3\frac{1}{2}$, sugar 6, proteids $\frac{3}{4}$. A week later he was given: fat 4, sugar 6, proteids 1, with disastrous results, vomiting and colic starting up in two days. He was then put onto: fat $3\frac{1}{2}$, sugar 6,

albuminoids ¾, did well and in a week was again tried on : fat 4, sugar 6, albuminoids 1, which he took well for a week, when again digestive disturbances forced us back to : fat 3, sugar 6, proteids ½, upon which he throve for six weeks, when he was promoted to : fat 4, sugar 7, proteids 1, doing well on this for two weeks, when, in hot weather, a slight diarrhœa necessitated a temporary return to : fat 3½, sugar 6, proteids ½. At seven months he had been for three weeks on : fat 4, sugar 7, proteids ¾, weighed 16 pounds, had two teeth, the first of which appeared one week before he was six months old, and notwithstanding the erratic course of his diet, his flesh was hard and firm and his general condition excellent. His subsequent history was uneventful, except that not until his eighteenth month was he able to take plain cow's milk.

Here was an infant, perfectly healthy at birth, but his digestive powers very early in life impaired by taking his own mother's breast-milk, poor in quality, on account of her nervous, hysterical temperament. As a result of these two factors, poor milk and impaired digestive power, he is weaned at two months, placed on low percentages of fat and proteids, the former of which is rapidly increased, the latter slowly and with difficulty, and he arrives at his 7th month in good general condition, it is true, but with weak proteid-digestive power, being able to take but ¾% of proteids, when he should be taking 1½ to 2%. The case illustrates the trouble which these infants with impaired digestive power entail, the necessity of constant watching and frequent change of formulæ, and also the great advantage of feeding exact percentages.

Diet in the Second Year.—Careful supervision of the child's diet during his second year should not be relaxed. Much trouble arises during this period from carelessness in the matter of food. The majority of children are allowed solid food too early and are generally over-fed. As already stated, weaning should be gradual, and the child accustomed to the change little by little.

The chief article of diet during this period must be plain milk. In some cases, however, it may be necessary to reduce the proteids, an element which we have already seen is apt to cause trouble in digestion. The same general principles laid down for the modification of cow's milk during the first year of life are also applicable in the next year, especially during the first half of it. We must remember that though we have a child fifteen or eighteen months old, he may have the digestive power of an infant six months old. The general condition, not the age alone, must be our guide and govern the amount and kind of food given.

At the end of the first year the starch-digesting power of the child is well developed. The amount of milk-sugar may therefore be reduced, and he may receive his carbohydrates in some other form.

Cereals may be added to the diet, preferably in the form of oat- or barley-jelly, given with an equal amount of milk. Bread and butter may also be given with milk. Broth, either chicken or mutton, may be taken for the midday meal. A baked potato may be given as early as the thirteenth or fourteenth month, an egg at about the sixteenth or seventeenth month. Only a few fruits can be taken at this period. Orange juice, well-cooked prunes and prune juice, a baked apple or apple sauce, a ripe peach or pear may be given with benefit, especially if there be any tendency to constipation.

The above régime is sufficient for the child until about the thirtieth month when we can again increase the variety by the addition of meat and vegetables other than potato, such as peas, beans, or lettuce. Meat, however, should not be allowed every day until the child is about four years old; it may be given two or three times a week. Children with "lithæmic" tendencies, and those with a diminished excretion of urea from other causes, do better on a limited amount of meat.

The practice of giving tea and coffee to children of all ages cannot be too strongly condemned. These stimulants should not be allowed until the sixteenth or eighteenth year, several years after the establishment of puberty with its nervous disturbance.]

CHAPTER V.

ACUTE SPECIFIC INFECTIOUS DISEASES: INTRODUCTORY.

Mortality due to the Acute Specific Infectious Diseases—Incubation Period—Prophylaxis—Complications and Sequelæ—General Remarks on Treatment: Nursing, Food, Drink, Alcohol—Hydrotherapeutic Treatment—Antipyretic Drugs—Treatment of Adynamia.

THE acute specific diseases are the cause of a very high **mortality** among children, and their sequelæ are the source of much ill-health. It appears desirable to indicate by the following statistics, extracted from the decennial summary, 1881–90, of the Registrar-General, their relative importance from this point of view.

ANNUAL MORTALITY PER 1,000,000 PERSONS LIVING FROM THE FOLLOWING ACUTE SPECIFIC DISEASES AT THE AGE-PERIODS STATED.

	0–5.	5–10.	10–15.	All Ages.
Whooping-cough	3,370	128	4	450
Measles	3,131	271	23	440
Scarlet Fever	1,669	762	153	334
Diphtheria[1]	690	424	100	163
Fever (Enteric, etc.)[2]	190	221	235	235
Small-pox	80	33	26	45

The main cause of this great excess in childhood is no doubt the absence of acquired immunity, but children are more liable than adults to contract certain infectious diseases—for example, mumps and, perhaps, scarlet fever and diphtheria. Certain others, as measles and whooping-cough, are commonly more severe in them. On the other hand, some, of which typhoid fever is an example, are as a rule less severe in childhood than at adult ages.

Infants at the breast enjoy a certain immunity. This is to be accounted for in some instances—as, for example, typhoid fever—by the rarity of their exposure to the ordinary mode of infection. A

[1] In the German Empire (*Mitt. a. d. K.-K. Gesundheitsamt*) the death-rate from diphtheria is much higher. In the age-period 1–15 years, the rate was 3,200 per million living in 1892, and 4,400 in 1893; but deaths attributed to croup were (no doubt, correctly) classed with diphtheria.

[2] "Typhus, Enteric Fever and Ill-defined Forms of Continued Fever."

similar explanation, however, is not applicable to some other diseases, of which measles may be quoted as an instance.

The diseases of this class possess certain peculiarities in common beyond the fact that all are communicable directly or indirectly, or in both ways. In their course three stages may be distinguished—the period of incubation, of fever, and of convalescence.

The **incubation period**—the interval between exposure to infection and the development of the characteristic symptoms—may be short, as in diphtheria, scarlet fever, influenza, cholera; or long, as in small-pox, measles, whooping-cough, mumps. The period of incubation is not constant in any disease, and the main variations are set out in the accompanying table. The period during which the disease is infectious, and more especially the period at which it is most infectious, is not the same in all. Thus measles, mumps, rubella, and whooping-cough are very infectious at an early stage, before the symptoms are characteristic; whereas scarlet fever and small-pox, though infectious at an early stage, become more dangerous during the later stages. On the whole, it may be said that the shorter the period of incubation the longer the subsequent period of infectiousness:

	Incubation Period (days).		Duration of Infection.		Period of Observation (days).
	Usual.	Extremes.	Beginning.	End.	
Small-pox	12	8–20	Prodromata	End of convalescence	21
Varicella	14	11–19	Onset	Ditto	20
Measles	9 or 10	4–14	Prodromata	3 weeks	15
Rubella	14–18	8–21	"	1 or 2 weeks	22
Scarlet Fever	1–3	1–7	Onset	Uncertain	7
Influenza	3	1–5	"	10 days	6
Whooping-cough	7–10	5–13	Prodromata	Uncertain	14
Diphtheria	2	1–7	Incubation	Undetermined	8
Enteric Fever	12–14	8–23	Onset	End of convalescence	24
Mumps	21	14–25	Prodromata	3 weeks	26

In the **prophylaxis** of acute specific diseases we have to consider the prevention of the spread of the disease to uninfected persons, and the prevention of complications in patients already suffering from it.

For the prevention of the spread of the disease regard must be had to the isolation of the patient throughout the period during which he is infectious, to the disinfection of excreta (stools, mucous and other discharges), and to the observation of susceptible persons who have been exposed to the infection. Early diagnosis is important, more especially in those diseases, such as measles, whooping-cough, and mumps, in which the patient is very infectious during the early stage. Before the appearance of the characteristic symptoms a positive diagnosis may often be impossible, unless there be an epidemic

in the district, or the child be known to have been exposed to infection. In all doubtful cases a guarded opinion should be given, and the child isolated and kept under observation. It is difficult in an ordinary private house to obtain effective isolation of scarlet fever or small-pox, and these diseases are of so serious a character that if a suitable fever hospital is available it is better for all parties that patients suffering from them should be removed to it. Diphtheria and enteric fever can be isolated effectively in a private house if a suitable room and intelligent nurses can be obtained; but in small houses and tenements a patient ought to be removed, if possible. Measles, whooping-cough, and mumps can be isolated in a private house in a suitable room; but many disappointments will be met with, owing not to the failure of isolation but to the fact that the infection has commonly been spread to other susceptible children during the prodromal stage. The period of time for which a person who is convalescent should be isolated varies in different diseases, and some general indications will be found in the table. In measles, rubella, whooping-cough, and mumps, efflux of time in itself diminishes the risk of infection; but in scarlet fever, small-pox, and diphtheria, this is not the case, since the infection may persist for long periods in the discharges of the skin and mucous surfaces, or survive in fomites, Every case must be decided after a full consideration of all the circumstances of the patient and his surroundings. The responsibility of disinfection after specific diseases—after those at least which in Great Britain must be notified—should be thrown upon the sanitary authority. During the fever the stools and urine, discharges, linen, and feeding and other utensils must be disinfected. For stools, urine, and discharges perchloride of mercury solution, 1 in 1,000, is to be preferred; for linen, a covered vessel containing 1 in 20 carbolic acid, in which the articles should be completely immersed. Utensils should be emptied and cleaned with soda solution, rinsed with perchloride solution and with tap water, and left to drain, or placed in a solution of boracic acid. Small articles are conveniently sterilized by boiling. The persons in charge of the patient should wear cotton gowns and aprons, attend scrupulously to personal cleanliness, and after handling the patient, his linen or discharges, should wash the hands, and then rinse them in a 1 in 2,000 perchloride.

Precautions of this nature serve also greatly to diminish the risk of many complications, especially broncho-pneumonia, which is the main cause of the large mortality produced by measles and whooping-cough. When it is necessary to nurse several children together, separate feeding utensils should be used for each. If one of the number develop broncho-pneumonia, it should be isolated from the others in a separate room.

If a child has been exposed to an infectious disease from which it is known not to have suffered, it is often of very great importance to decide the length of time during which it must be kept under observation before it can be declared to have escaped the infection. The period should exceed the longest known period of incubation of the disease in question, and care must be taken to ascertain at its termination that the patient is free from all prodromal symptoms.

Complications and Sequelæ.—The number and variety of the complications and the sequelæ which may arise in connection with the acute specific febrile diseases is immense. There is no organ or system of the body which may not become affected, and the nature of the complication is determined in part by the special manifestations of the infection, and in part by the constitution of the individual; that is to say, the breakdown is most likely to occur, other things being equal, at the point of least resistance. Among the most frequent and dangerous complications are affections of the respiratory system, and local inflammations such as otitis or ophthalmia, due to secondary infection. They are therefore accidental, and to a greater or less degree in individual cases, preventable complications, and the reader is referred to the pages in which the diseases of the several organs are considered. Gastro-enteritis and colitis also, by which the acute specific diseases are very apt to be complicated in childhood, are best studied in connection with these disorders when due to other causes.

Any acute febrile disorder may be complicated by albuminuria, and the acute specific diseases afford no exception to the rule. The condition will be discussed subsequently, when the diseases of the kidney come to be dealt with. But it must be observed here that the presence of albumen in the urine during the febrile stage is of very different significance in prognosis from its appearance or persistence after the febrile movement due to the specific infection has passed away—in the one case it may be attributed to the transient effects of toxæmia on the kidneys; in the other to local disease of these organs.

Mental Disorders.—The delirium which attends the febrile stage does not call for special discussion here, but after defervescence various disordered mental states may cause much anxiety. The most common is a condition of mental apathy, which may amount to actual dementia. Its cause is to be found in the condition of defective general nutrition and anæmia produced by the disease, and in the effects of severe or long-continued toxæmia on the nutrition of the brain in particular. In other cases attacks of delirium and maniacal excitement recur frequently during convalescence. Such attacks are specially prone to come on after meals or at night; that is to say, at times when there is a physiological tendency to cerebral

anæmia, and they are to be attributed to an exaggeration of this tendency, which produces the greater effect owing to the existence of general anæmia. The occurrence of nocturnal delirium may give rise to apprehensions of relapse of the disease, but the temperature is usually normal or subnormal. Apathy or dementia is most often a sequela of typhoid fever or measles, but it may occur after other acute febrile diseases. After influenza, an allied condition described under the term somnolence is observed occasionally in children and even in infants, as in adults. Sometimes, especially after typhoid fever, delirium and apathy alternate, the patient being apathetic by day and delirious by night. Mania and maniacal excitement are observed more often as a sequela of scarlet fever than of the other febrile diseases,[1] but are not unknown after others, especially typhoid fever. Occasionally a conspicuous feature of the apathetic state is mutism and in some instances a condition of aphasia persists for some weeks after the patient has made great improvement in other respects. This is observed most often after typhoid fever. The child may be able to see and hear, even to write, but is unable to utter a word. The power of speech returns as a rule at first slowly and then very rapidly, so that complete aphasia may in a few days be succeeded by free speech.

In the treatment of apathy, nocturnal delirium, or dementia after acute febrile diseases, the main indication is to improve nutrition by suitable diet and tonics, and to diminish the anæmia by the administration of iron salts, of which the perchloride is the best if it can be borne by the stomach. Nocturnal delirium can usually be controlled by moderate doses of potassium bromide given during the afternoon and evening, but a dose of chloral may be necessary in some cases to procure sleep.

Nervous disorders are among the most serious, though least frequent, complications. The occurrence of hemiplegia is discussed elsewhere, but generalized nervous disorders also occur. After small-pox, measles, scarlet fever, typhoid fever, whooping-cough, influenza, and also in association with erysipelas, acute pneumonia, and perhaps ague, cases are occasionally met with which present symptoms of widely diffused nervous affections probably allied pathologically to diphtherial paralysis. They may be classified as follows: (1) Cases of "extensive, ascending, diffuse, or disseminated" paralysis, resembling diphtherial paralysis. (2) Cases in which, with some symptoms similar to those of the preceding group, the most prominent symptom is incoördination. (3) Acute disseminated myelitis. (4) Cases presenting at a later date symptoms resembling disseminated sclerosis (false disseminated sclerosis).

In cases belonging to the first and second class the paralytic or

[1] *Conf.* Mercier, *Brit. Med. Journ.*, 1883, vol. ii., p. 630.

ataxic symptoms are observed usually first when convalescence has already commenced, but in those in which disseminated myelitis developed, or in which symptoms resembling disseminated sclerosis have subsequently appeared, convulsions have occurred during the febrile stage, or stupor or somnolence has been noticed during that stage. The patient on recovering consciousness is unable to speak, and is found to be suffering from extensive paralysis or paresis, or from ataxia. From this condition he may recover completely, or he may develop symptoms resembling those of disseminated sclerosis, but with this difference, that the disease is not progressive but rather regressive. In the cases fatal at an early stage, the changes in the central nervous system appear to have been mainly or primarily vascular; and it seems reasonable to assume that in those cases in which complete recovery takes place at an early date the symptoms are due to vascular derangement. The recovery from diphtherial palsy is not always complete, and in a few cases it has been known to be followed by this condition of "false disseminated sclerosis."

The observation lends support to the view that cases of the kind here under consideration, whether instances of transient paralysis, of ataxy, or examples of a train of symptoms resembling disseminated sclerosis of the cord or hemispheres, are due to the action on the nervous centres of soluble toxins circulating in the blood.[1]

Muscular atrophy resembling that produced by acute polio-myelitis, and due probably to a lesion in the same region, has been observed occasionally as a sequela of acute specific diseases. Complete recovery has in some cases occurred, but in others the palsy has been permanent.

General Remarks on Treatment.—In treating a case of proved or suspected infectious fever, the first step is to isolate the patient in a well ventilated room, from which carpets, heavy curtains, and superfluous articles have been removed. This is recommended in the interests not only of others, since such articles are difficult to disinfect, but also of the patient, since they are a cause of dust, which may be the source of secondary infection.

Nursing.—A trustworthy trained nurse should, if possible, be obtained. She should be competent not only to attend to the comfort and cleanliness of the patient, and to the regular administration of food and drugs, but also to note the general progress of the case, and the time of the supervention of new symptoms, for the information of the medical attendant. She should be made responsible for the ventilation of the room, the temperature of which should be kept

[1] This subject will be found more fully discussed in the papers by Marie (*Prog. Med.*, 1884, No. 15, *et seq.*); by Whipham and Myers (*Clin. Soc. Trans.*, xix., p. 164); by Barlow (*Med. Chi. Trans.*, lxx., p. 77); and by the present writer (*Ibid.*, lxxvii., p. 57).

about 56° F. or, in influenza or measles, 58° F. She should also attend to the proper treatment, with a view to their disinfection, of excreta and all discharges, or objects soiled with them, such as linen, and feeding and other utensils.

Food.—The main object in the symptomatic treatment of fevers is to maintain the energy and nutrition of the nervous and muscular tissues. The difficulty is the greater the more intense the infection, the less the power of resistance of the individual, and the higher the fever. The probable duration of the specific process will influence treatment, since the task is more difficult in fevers of long duration, such as typhoid fever and variola. The diet should contain appropriate quantities of proteid, carbohydrate, and fat. There is often a disposition to give too small a quantity of carbohydrate food; it must be remembered that carbohydrates added to the diet increase the amount of proteids assimilated, and diminish the loss. Gelatine also is an economizer of proteid waste. In arranging a diet, especially in an illness which is likely to be prolonged, it is important to avoid monotony. In children the staple article of diet should be milk, which may be modified in various ways, as by dilution with barley water or by peptonization. The value of peptonized milk, diluted if necessary, is very great in severe cases. The amount of carbohydrate in the diet may best be increased by giving wheat, oat, or barley gruel, which is more palatable if made with milk and sweetened, and there is no objection to the use of lemon or other simple flavorings. Soups, if carefully made and well flavored with vegetables passed through a sieve, afford a useful variety often much appreciated by the patient, and one which has a favorable influence on the bowels. Beef-tea should be banished; even when well made it contains little nutriment, and the ordinary product is poisonous.

Drink.—The person in charge of the child should be impressed with the fact that milk is a food and not a beverage. Children with fever often suffer intensely from thirst, and if this fact be not realized are very apt to be given far too much milk. Barley water, which may be flavored with lemon, fresh lemonade, seltzer water, and other simple beverages should be allowed. (*Fresh Lemonade.*—Rub two or three lumps of white sugar on the clean rind of a lemon, squeeze out the juice and remove pips and shreds; place together in a jug with a bottle of soda-water or an equal quantity of boiled (cold) water. Appendix.) The drink may be given at the ordinary temperature of the room. If the thirst be associated with stomatitis or pharyngitis, small sips of iced water may be given, or in children old enough to be trusted morsels of ice may be sucked.

The danger of over-feeding must be avoided by prescribing the quantity of food, and directing an appropriate amount to be given at regular intervals. As a general rule, the indication is to give

small quantities at short intervals. It is difficult to lay down any rules as to quantity, as a judgment must be formed in each case from the indications afforded by the tongue and the condition of the digestive organs; but if a child between one and two years old be taking daily a pint of milk, in various forms, it may be considered to be having quite as much as is desirable.

Alcohol.—The routine resort to alcohol is to be condemned, but it is a valuable drug when a stimulant is required. For this purpose good brandy is probably the best. The quantity to be given should be precisely stated.

Antipyretic Treatment.—Since the febrile state is the natural reaction of the organism to infection, it may be assumed to possess a useful function; and it might therefore be argued that attempts to reduce the temperature are irrational. The theory, so far as it tends to obviate unnecessary interference, may have a wholesome influence; but the reaction may be excessive, and, apart from the question of hyperpyrexia, may call for treatment. A rise of temperature is attended by alterations in the metabolism of the tissues, an increase of oxidation, and an arrest of digestive secretions. A moderately high temperature, which does not produce a marked effect on the heart or nervous system, and is not long lasting, does not call for antipyretic treatment. On the other hand, if, owing to the height to which the temperature reaches or the length of time for which it remains elevated, there is a marked diminution in the energy of the nervous system or heart, then it will be necessary to take means to reduce it.

Hydrotherapy.—The application of cold to the surface by baths or wet packs is, as a general rule, the most efficient and safe method for reducing temperature. The antipyretic effect is produced partly by the direct abstraction of heat, partly through the peripheral nerves, which, when stimulated by the cold, produce an effect on the nervous centres, diminishing coma and delirium, improving the action of the heart and the respiration, and stimulating the heat-regulating mechanism. The effect of the application of water to the surface at a temperature lower than that of the body in fever is very considerable, and is especially marked in children. Thus Eröss[1] found that in infants with temperature at about 104° F., a bath for ten minutes at 95° F. caused a fall of 3.7° to 9° F., the greater part of which took place usually during the bath, but part during the hour or two after; then the temperature began to rise again, but did not reach its former height for three, four, or even ten hours.[2] The main objection to the bath is that its action, even when the water is luke-

[1] *Jahrb. f. Kinderhlkde.*, Bd. xxxii., s. 83.

[2] Eröss (*loc. cit.*) found also that even in healthy infants a bath at 80° to 86° F. for 10 minutes produced a fall of 3° to 5.7° F., the normal temperature not being regained until three to five hours later.

warm, is apt to be too energetic. Although it is true, as a general proposition, that the lower the temperature of the bath and the longer it is applied the greater the effect on the temperature of the body, yet it is not easy to foresee in children the degree of the effect, owing to the rapidity with which in them the temperature may be reduced. The lukewarm bath is, however, a very valuable therapeutic means if it be remembered that the effects which it produces must be watched carefully. The cold bath, which has a very energetic action, may be of use in those rare cases in which, during the early stage of a fever, the condition has become dangerous, owing to the intensity of the cerebral symptoms. Such a bath not only reduces the temperature but stimulates the nervous system and the heart. A similar effect is more safely obtained by placing the child in a warm bath and pouring cool or cold water on to the head and shoulders. If a suitable bath is not available, or should its use for any reason appear inadvisable, the temperature may be reduced 2° to 3° F. by stripping the patient and applying to the surface a sheet wrung out of water at 55° to 60° F. This sheet is removed every five minutes for four or five times, the last sheet being used as a pack, in which the patient is kept covered with blankets for twenty minutes or half an hour.

Baths: Warm Bath and Pack.—The bath should be large in proportion to the size of the child, and a large quantity of water (at first at the temperature of 95° F.) should be used. During the bath, which should last twenty minutes, the temperature of the water should be raised to 104° F. or 105° F. by the careful addition of hot water. The child should be kept immersed up to the neck, and a blanket should be thrown loosely over the bath, and held around the neck. Meanwhile, a bed is to be prepared thus:—Turn down the bed-clothes, and put a blanket on the bed, so that it projects a little over the foot. Immediately before the child is to be taken out of the bath, a fairly thick sheet, thoroughly wrung out of hot water, is placed over the blanket. The patient is now lifted out of the bath, and laid on the sheet, in which it is tightly wrapped up, with the arms inside; the part of the sheet which projects beyond the feet is folded and tucked firmly under the feet. In adjusting the sheet, care must be taken to avoid creases, and to tuck it in firmly around the neck. The blanket is now folded around the patient, great care being taken to avoid any looseness or irregularity by which air could enter. The bed-clothes are now pulled down, and tucked in firmly at the foot, sides, and neck, so as to exclude the air. Profuse perspiration commonly begins in a short time. The patient should remain in the pack about an hour. The pack is then undone, and the patient quickly rubbed down with a warm, rough towel, put into a second bed (which has been well warmed), if such be available, and covered

with plenty of light, warm bed-clothing. The shift from the pack to the bed must be done as rapidly as possible. In mild weather the window may be open while the patient is in the pack, but draughts should be avoided. (Jürgensen.) [Appendix.]

Antipyretic drugs are now much less used than was the case formerly. The reduction of temperature effected by quinine lasts longer than that produced by other antipyretic drugs, but it is apt to derange the stomach. If the sulphate is used, it should be given every six or eight hours in doses of about 1 grain for each year of life. Quinine tannate in powder is more readily taken, since its taste is less bitter, but the dose must be double that of the sulphate. Quinine may also be given by subcutaneous injection, and the best salts for the purpose are the hydrochlorate or hydrobromate. Antipyrin and antifebrin produce a depression of temperature which is of shorter duration, but the former in particular has a sedative effect, which is often beneficial.

Adynamia, characterized by great nervous depression and cardiac failure, is the cause of death in many severe cases. Warm baths of short duration, with, if the temperature is high, cool affusions to the head, should be employed and their effects watched, or an ice-cap may be applied to the head. The failing heart is stimulated by these means. For this purpose alcohol is useless, if not injurious, except perhaps in severe diphtheria and fevers partaking of the septic type. Digitalis is of somewhat uncertain advantage; it is best given in one or two full doses, and may be followed or replaced by strophanthus. Failing heart, accompanied by coldness and blueness of the extremities, dyspnœa and pulmonary œdema, should be combated by small doses of sodium nitrite ($\frac{1}{10}$ to $\frac{1}{4}$ gr.) or nitro-glycerine ($\frac{1}{500}$ to $\frac{1}{100}$ gr.).[1] Strychnine is probably the most valuable drug in the treatment of the general symptoms of adynamia; an infant of a year old may have a hypodermic injection of $\frac{1}{100}$ grain three or four times a day, and the dose may be increased considerably if necessary. Hypodermic injections of camphor are much used in Germany, and of caffeine in France, but the latter is unsuitable if there are symptoms of cerebral irritation. Of camphor 5 to 15 minims of a 10-per-cent. solution in oil may be given to children of five or six years, even as frequently as every hour, the indication being failure of the pulse.

It may also be given as follows [Appendix]:

Elixir Camphoræ (Martindale & Westcott)	
Spirit of Camphor	℥x
Syrup	℥v
Distilled Water	℥j
Contains camphor gr. iv in	℥j.

[1] Possibly erythrol tetranitrate might be more efficacious, since its action is more prolonged.

Caffeine may be administered by prescribing strong black coffee, which, however, is not suitable for young children. For subcutaneous injection, caffeine should be prescribed in a watery solution of sodium benzoate. The dose for a child of two years should be 2 grains.

It is prepared as follows [Appendix] :

In water ℥j dissolve sodium salicylate gr. xvj or sodium benzoate gr. xx, and add caffeine gr. xx. Sterilize by boiling for 15 minutes. Gr. j in ♏ iij.

CHAPTER VI.

ACUTE SPECIFIC INFECTIOUS DISEASES (*continued*).

Small-pox—Vaccination—Symptoms and Treatment of Small-pox—Varicella—Measles—Rubella—Scarlet Fever.

Small-pox, which used to be an almost inevitable accident of human life, has now, in vaccinated communities, become a rare disease, especially in childhood. For example, of 1,117 cases admitted into the hospitals of the Metropolitan Asylums Board in 1894, 127 were children under seven years of age, and 178 children from seven to fourteen years. Of the 127 children under seven, 111 had not been vaccinated, and all the deaths (thirty-five) at this age-period occurred among them. Children unprotected by vaccination are very liable to small-pox, and their mortality is high. Of the unvaccinated persons who died in England and Wales, in 1881–92, 38.9 per cent. were under five years old, and 72.9 per cent. under twenty years. Of the vaccinated persons who died of small-pox (including in this number those as to whom no statement as to vaccination was made) 16.8 per cent. were under five years, and 38.9 per cent. under twenty years. The relative rarity of small-pox in childhood in Great Britain is undoubtedly due to the fact that the majority of children are protected by vaccination in infancy, while re-vaccination, necessary to renew protection, which diminishes after eight to ten years of age, is not so universally practiced.

Vaccination.—When vaccinia runs a normal course a small papule is observable at the points of insertion about thirty-six hours after vaccination; this grows larger, and about the fifth day shows a vesicular top with a depressed centre. The vesicle enlarges, and becomes surrounded by a red, thickened edge. It attains its characteristic stage on the eighth day, when it is a large flat, umbilicated vesicle of an opaque white or opalescent color. For the next two days it enlarges slightly, and becomes surrounded by a wide areola of inflamed skin. On the eleventh or twelfth day the stage of regression begins; the areola becomes less marked, the vesicle flatter, its contents more opaque. By the fourteenth or fifteenth day it is converted into a scab, which is detached after a week or two weeks more, leaving the characteristic depressed cribriform scar. At the

height of the vaccinia there is usually some enlargement of the axillary glands, which are tender.[1] Suppuration occurring under the scar is due to secondary infection by pyogenic organisms, and should be treated locally by antiseptic applications.

The degree of immunity conferred by vaccination varies, but when efficiently performed it is almost complete, and the individual, if exposed to the infection of small-pox, either escapes or, if he contract the disease, suffers from it in a more or less mild form—modified small-pox. The duration of the protection afforded by primary vaccination in infancy is uncertain, and re-vaccination is necessary at the age of nine to ten years. During an epidemic re-vaccination should be practiced at the age of seven years.

In presence of an epidemic there should be no hesitation in vaccinating the youngest infant. Under other circumstances, vaccination should be performed between the ages of three and six months. It should not, under ordinary circumstances, be performed if the infant is suffering from any pyogenic form of dermatitis, nor in syphilitic children unless otherwise in good condition, nor if there be any known source of erysipelatous contagion. A certain amount of judgment must be exercised in the case of marasmic children; as a rule they bear it well, and it should not be deferred if the disease be epidemic. As the vaccination vesicle is attended by a good deal of itching during the stage of areolation, the child is apt to scratch or rub, and so break the vesicle, which then usually becomes the seat of suppuration. Under such circumstances care should be taken to prevent secondary inoculations on other parts of the body by the finger-nails or clothes. The application of boracic ointment is often useful if the pock be ruptured. Various rashes may occur during the time of the maturation of the vaccine vesicle. In my own experience the most common has been a discrete papular rash, some of the papules showing slight vesiculation. Irregular erythematous rashes, which sometimes leave a slight yellow pigmentation, may also be observed; but the most frequent rash would appear to be roseola, coming out from the eight to the eleventh day after vaccination. All these rashes are rare, and the rarest general affection is true generalized vaccinia. When vaccinia runs its customary localized course it causes very little disturbance of the general health, and elevation of temperature is not a constant symptom. Re-vaccination is often attended by more widespread local reaction and more general disturbance. The use of impure vaccine (from contaminated vesicles,

[1] When small-pox was inoculated a papule appeared on the second day at the point of inoculation; this had become a vesicle on the fourth and a pustule on the eighth, on which day the patient had rigors, much local swelling with adenitis, and fever. The ordinary small-pox eruption appeared on the eleventh day. The course of the local lesion after vaccination and variolation respectively may be studied in the beautiful series of drawings reproduced in the *British Medical Journal* of May 23, 1896.

or contaminated during collection) has caused septicæmia. Such accidents may be prevented by the use of calf lymph prepared with glycerine. Erysipelas may ensue on vaccination under unfavorable circumstances. The vaccine vesicle, on about the tenth to the fourteenth day, instead of disappearing, may become the seat of deep ulceration, owing probably to secondary inoculation under unfavorable hygienic surroundings. All such sequelæ are very rare. In a few instances syphilis has been inoculated with vaccinia; in many more instances in which this has been supposed to have happened the syphilis from which the infant has suffered after vaccination has been congenital, and no case can be admitted to be an example of syphilis due to inoculation at vaccination unless a primary chancre be discoverable.

[The glycerinated lymph is put up in sealed glass tubes and in this country is furnished by the city laboratory. It is prepared with great care, and is far superior to the old-fashioned "points." Its more general use is greatly to be desired to prevent unnecessary infection. Strict antiseptic precautions should be taken in the vaccination, the physician's hands and patient's skin at the seat of the operation being made as nearly aseptic as possible. A sterile knife should be used for making the incisions and these should not be deep enough to draw blood. One incision is sufficient for an infant, two for a primary vaccination in a child under puberty, and three for any re-vaccination. The only dressing required is a piece of soft linen pinned or sewn to the inner side of the sleeve, or underdrawers when the vaccination is on the leg, to prevent rubbing or chafing of the affected part.]

The *incubation period* of small-pox is usually twelve days, not infrequently a day more or a day less; sometimes it is as short as nine days or as long as twenty. The period of observation should therefore be three weeks. The infection is greatest during the height of the eruption. It is much less active during the initial stage so that the risk of infecting others may be diminished by isolating a patient at the beginning of the eruptive period: but all persons who have been exposed should be vaccinated (or re-vaccinated) and kept under observation. Infection remains until the scabs have cleared off completely. It may be carried by fomites (and preserved long in them) and in the clothes and hair of persons in attendance on a patient.

The active agent of the infection is believed to be a small bacillus which has been shown by Klein and Copeman to be present in the lymph and the tissues about the pock about the fifth day in variola in man, and in vaccinia both in man and the calf. Before maturation it ceases to be discoverable, owing perhaps to the formation of spores.

The *onset*, which is sudden, is marked by chilliness, or by convulsions, which may be repeated several times during the stage of inva-

sion. The temperature rises quickly to about 104° F., and throughout this stage the child suffers from severe backache, headache, and pain in the limbs, and from nausea or vomiting. "Initial rashes," which occur in about one-sixth of all cases, usually appear on the second day, and commonly resemble, often very closely, those of measles or of scarlet fever. These rashes, however, are confined usually to the lower part of the abdomen, the axillæ, and the sides of the chest, or the extensor aspects of the limbs. Even in modified small-pox the symptoms during this stage may be very severe, but as a general rule they are proportionate to the subsequent eruption; that is, they are milder and longer lasting in cases in which the eruption is discrete; shorter and more severe in confluent small-pox.

The *eruption* in discrete small-pox begins usually on the fourth day. On the forehead, near the margin of the hairy scalp, and on the wrists, small, slightly elevated papules appear, and are very perceptible to the touch, owing to their firm, "shotty" character. A little later they come out on the face, limbs, and scantily on the trunk. The eruption may affect also the mucous membrane (mouth, pharynx, larynx). In about forty-eight hours after its first appearance each papule has become an umbilicated vesicle, and within it, in about forty-eight hours more (seventh to eighth day), suppuration has begun. The umbilication disappears, and the pustule thus formed is surrounded by an areola of red swollen skin.

The *temperature*, which rises at the onset and attains its maximum (104° to 105° F.) during the second and third days of the stage of invasion, declines rapidly as the eruption comes out. When suppuration begins, it rises again and remains elevated, with a morning fall and evening rise. During this stage of maturation the maximum is touched on the ninth or tenth day, after which each evening rise is to a point lower than on the previous day. This decline attends the drying of the pustules into scabs or crusts, which begin to be detached seven or eight days after the papules appeared (about the twelfth day of the disease). Sydenham's observation, that the danger to life is in proportion to the severity of the eruption on the face, has been confirmed by all experience. In the confluent form the papules, which appear early (third day), are very numerous on the face, and also on the hands and feet, and the vesicles and pustules run together into large blebs. The crusts are large, and ulceration is apt to occur beneath them. The symptoms during the stage of invasion are more severe, and the remission of temperature on the appearance of the eruption is less marked; during the stage of maturation it rises to 104° F., or higher, there is extreme restlessness or delirium, and often diarrhœa. The eruption is usually present in the pharynx and larynx (so that the patient is hoarse), and often in in the mouth. The most usual cause of death is failure of the heart

and collapse of the nervous system, evidenced by delirium giving way to coma. Of hæmorrhagic small-pox, which is rarer in children than in adults (of Osler's twenty-seven cases, three only were in children under ten) two types must be distinguished—(1) *Purpura variolosa*, a fulminating form of small-pox, in which the initial symptoms are of great intensity. There is an initial purpuric rash, hæmorrhages into the conjunctivæ and from the mucous surfaces, and death at an early date, often before the characteristic eruption appears. (2) *Variola pustulosa hæmorrhagica*, in which blood is effused into the vesicles or pustules, and bleeding may take place from mucous surfaces. The earlier the date at which the hæmorrhage begins, the worse the prognosis, and this form is very fatal. Cases occur, however, occasionally in which, during the vesicular stage, hæmorrhage takes place into the pocks, but in which, nevertheless, the course is favorable, the vesicles drying up and the disease aborting.

Varioloid, small-pox modified by vaccination, is seldom seen in early childhood, since children efficiently vaccinated, as a rule, escape infection altogether, the initial symptoms, usually comparatively mild, though the pain in the head and back may be very distressing, are sometimes severe, but the papular eruption is scanty, and with its appearance the temperature drops rapidly and does not again rise. Vesiculation and maturation are completed rapidly, and the patient enters on convalescence early.

In childhood the most frequent and serious *complications* of small-pox are (1) broncho-pneumonia, which is a contributory cause of death in most fatal cases; (2) laryngitis, which may cause death by producing œdema glottidis, ulceration of cartilages, or indirectly by blunting laryngeal sensibility, so that food is allowed to enter the air passages; and (3) diarrhœa. Otitis media is a frequent secondary complication. Conjunctivitis, sometimes severe and purulent, leading to keratitis and perforation, is not uncommon.

Prognosis.—Unmodified small-pox is extremely fatal in young children, and few infants recover from it. Death, as a rule, occurs either in the early stage from the intensity of the disturbance of the nervous system or at the height of maturation, and is then usually hastened or determined by laryngitis or broncho-pneumonia.

In the **treatment** of small-pox, precautions against the spread of the infection must be rigidly enforced (*vide ante*). For the relief of the severe backache and headache of the initial stage small doses of opium frequently repeated, or two or three doses of phenacetin, are useful. High fever may be treated by baths. Many remedies have been tried for the prevention of pitting, but Sydenham, one of the earliest, and Osler, one of most recent writers on the subject, agree that local remedies have no influence, or are actually injurious.

During the papular stage cold, applied by means of a face mask kept moist with water, to which an antiseptic (carbolic or perchloride) is added, is grateful to the patient, and probably tends to check the deepening of the pocks, and should be continued later, since it tends to check the formation of hard, dense crusts. When crusts have formed they should be kept constantly soft by the application of vaseline, glycerine, or an ointment made with equal parts of lanoline and olive oil, to which some antiseptic, such as boric acid, should be added. The patient should have also one or two baths a day, for the removal of epithelial *débris* and crusts. The baths should be medicated with thymol, carbolic, or other antiseptic, and carbolic soap should be used. Stomatitis should be treated with antiseptic mouth washes and creams. The conjunctivæ should be inspected, and on the signs of commencing inflammation should be thoroughly washed with antiseptic lotion three or four times a day, and if the eyelids are closed by swelling this precaution should be at once adopted. For severe diarrhœa some preparation of opium, the tincture of paregoric in small doses frequently repeated, should be used. The theory that the pustular stage is rendered less severe and subsequent pitting less deep by nursing the patient in a red light seems to be well grounded. It may be carried out by hanging the windows with one thickness each of red and yellow photographic calico.

Varicella, a disease from which few children escape, is therefore seldom seen in adults. It is characterized by a vesicular eruption. The incubation period is usually fourteen days, but may be a day or two less, or four or five days more. The disease is infectious as soon as the rash appears, and a convalescent patient may convey to others the infection, which is retained also in fomites.

The disease is usually extremely mild. At the onset the patient complains of malaise and chilliness, and there is a very slight elevation of temperature. The eruption usually begins within the first twenty-four hours on the face and neck. It consists of scattered papules, which in a few hours become converted into vesicles. The vesicles are ovoid and not, as a rule, umbilicated. On the third or fourth day they begin to shrivel, but before this the majority of them have become purulent. The eruption often appears in distinct crops upon the face, trunk, and limbs, so that those at the upper part may already be scabbed while those on the limbs are vesicular. They vary in number, from half a dozen to hundreds. As a rule, the scars left are very superficial; but if the child is allowed to scratch in the pustular stage, extensive ulceration, scabbing, and scarring may be produced, and in marasmic children large ecthymatous patches, or bullæ, may form. Occasionally hæmorrhage takes place into the vesicles, or into the surrounding skin, which is usually healthy; or bleeding from the mucous membrane may occur. A rare complica-

tion is gangrene around the vesicles. The eruption may appear, usually at a very early stage, on the mucous membrane of the mouth (soft palate, cheeks, gums) of the larynx, of the vulva, the conjunctiva, and in the external auditory meatus. In the mouth, where they are most often seen, the vesicles rupture early, leaving superficial circular erosions, with which may be associated severe stomatitis. Albuminuria is present during the fever in a small proportion of cases, sometimes persists for a fortnight, and is occasionally accompanied by anasarca. Arthritis has been known to occur.

The **prognosis** is good. Uncomplicated varicella is seldom or never fatal. Varicella gangrenosa is a serious disease, and in cases complicated by albuminuria may cause death.

The **diagnosis** is usually easy, but cases occur occasionally which resemble variola, and the mistake has been made. The eruption in varicella, however, runs its course very rapidly; redness, with at most very slight thickening of the spot of skin to be the seat of the vesicle, is succeeded in a few hours by the effusion of clear liquid into the epidermis, forming a watery pock with a very thin wall. It does not involve the true skin, and if umbilication is to be observed, it is present only in a few vesicles. When, as is often the case, the vesicles are very numerous, the mildness of the general symptoms will put small-pox out of the question, and the slight character of the symptoms at the period of invasion will afford corroborative evidence, for it will be remembered that even in modified small-pox the initial symptoms are usually severe. Impetigo has been mistaken for varicella, but a close examination of the lesions and of their mode of development will prevent such an error. Rather more difficulty may be experienced in distinguishing pemphigus from varicella, but the course of the disease will quickly clear up any doubt.

The **treatment** must be conducted on general principles. The child should be kept in bed until the temperature has fallen. Scarring seldom occurs if attention be paid to cleanliness and to the prevention of scratching, which may lead to the inoculation of the vesicles with pyogenic organisms. A mild antiseptic, such as boracic ointment, is to be recommended as an application to any pocks which show a tendency to suppurate.

Measles is an acute infectious fever, characterized by a prolonged stage of prodromal catarrh and a peculiar eruption.

The interval between exposure to infection and the appearance of the rash is usually fourteen days, or one day more or less, but it may be as long as eighteen or as short as seven days. The infection is very active during the primary stage; that is, for three or four days before the rash appears, and probably not less so during the whole acute attack. It diminishes rapidly during convalescence, and has ceased at the end of three weeks from the appearance of the rash.

PLATE I.

Fig. I.

Fig. II.

Fig. III.

Fig. IV.

The Pathognomonic Sign of Measles (Koplik's Spots).

Fig. 1.—The discrete measles spots on the buccal or labial mucous membrane, showing the isolated rose-red spot, with the minute bluish-white centre, on the normally colored mucous membrane.

Fig. 2.—Shows the partially diffuse eruption on the mucous membrane of the cheeks and lips; patches of pale pink interspersed among rose-red patches, the latter showing numerous pale bluish-white spots.

Fig. 3.—The appearance of the buccal or labial mucous membrane when the measles spots completely coalesce and give a diffuse redness, with the myriads of bluish-white specks. The exanthema on the skin is at this time generally fully developed.

Fig. 4.—Aphthous stomatitis apt to be mistaken for measles spots. Mucous membrane normal in hue. Minute *yellow points* are surrounded by a red area. Always discrete.

It may be conveyed by fomites. When inoculated the incubation period is less than ten days.

The severity of measles varies in different epidemics, but more important factors are: (1) the general hygienic surroundings of the patient—when these are bad the chance of a severe attack is increased; and (2) the age of the patient—the death-rate is very high (even over 50 per cent. in some epidemics) in children under two years old. It is still high from two to four years, but declines rapidly after that age. Canon and Pielicke have described a small bacillus as present in the blood in measles, and their results appear to be confirmed by Czajkowski, who found a similar bacillus in the blood of fifty-six cases examined for this purpose.[1]

The *prodromal*, primary, or catarrhal stage endures usually between three and four days; it may be very much shorter, even only a few hours, and is extended occasionally to seven or eight days. The child is chilly, loses appetite, and begins to suffer from coryza and photophobia. Complaint is made often of headache and, within the first day usually, cough becomes troublesome. The tongue is furred, the pharynx hyperæmic, the soft palate sometimes covered by a punctiform rash. [An eruption also appears on the buccal mucous membrane, regarded by Koplik as pathognomonic of beginning measles. This eruption, called the *enanthem*, appears very early, before the exanthem, is seen on the buccal mucous membrane and inside of the lips, consists of small irregular spots of a bright red color in the centre of which is noted in strong daylight a minute bluish-white speck. Koplik regards it of the greatest diagnostic value early in the disease before the appearance of the skin eruption. Plate I.] The temperature rises rapidly, attaining 103° to 104° F. on the first day; it then falls 1° or 2°, but rises again on the fourth day just before the rash appears. This usually comes out rapidly often during the night, affecting first the cheeks, forehead, and the skin behind the ears; then the neck, the trunk, and, lastly, the limbs. The *rash* consists of discrete papules, which enlarge, increase in number, and become arranged in crescentic patches. The characteristics of the rash are best seen on the face and neck. In these parts the patches have a dusky or purple tinge, are distinctly raised and hard, and are attended by more or less œdema of the surrounding skin. Miliary vesicles often form, and petechiæ may appear even in mild cases. In malignant cases there may be a good deal of hæmorrhage into the spots. On the trunk the rash is often less distinctly papular, and may be no more than a dusky mottling. The rash continues to spread for twenty-four or forty-eight hours, or a little longer, and during this time the temperature remains high, reaching perhaps 104.5° F. on the second day of the rash. The coryza, cough, pho-

[1] *Cent. f. Bakt. u. Par.*, Band xviii., Num. 17, 18.

tophobia, and other general symptoms remain severe for about the same period. The rash in each region begins to fade in about thirty-six hours, so that it may be disappearing on the face while still coming out on the limbs. If there be no complication all the symptoms begin to improve so soon as the rash ceases to extend. The temperature falls rapidly, though not as a rule by a distinct crisis, and reaches normal on the seventh or eighth day from the commencement of the illness. The rash is attended by a good deal of hyperæmia, so that the color can be almost completely discharged when at its height, but a slight yellow stain may remain after the rash has faded. Fine branny desquamation, most marked on the trunk and lower extremities, but often very inconspicuous, follows after the disappearance of the rash.

The most important **complications** are those of the respiratory system. Bronchitis, which is rather a symptom than a complication, predisposes to the occurrence of broncho-pneumonia in the manner indicated in the chapter on this disease. This complication is responsible for the greater part of the high death-rate. Lobar pneumonia is less common, but the specific poison of measles appears to be able to produce a special form. Laryngitis, frequent in some epidemics, may cause œdema of the glottis. [It may be membranous and, developing at the height of the disease, is generally due to the streptococcus; but when it develops at a later period, it is usually due to the diphtheria bacillus. Holt states that it occurs more frequently as a complication of measles than of scarlet fever, and that as a cause of death in older children, it ranks next to pneumonia.] Stomatitis, which may lead to severe ulceration, or cancrum oris may occur in debilitated infants, as may also ulcerative vulvitis. Otitis media, which is secondary to pharyngitis, is a common complication and may lead to mastoid suppuration, to perforation, and to permanent loss or dulling of the sense of hearing. Conjunctival catarrh occasionally runs on into suppuration, and tinea ciliaris is a not uncommon sequel. Severe diarrhœa, due in many cases, at least, to membranous colitis, occurs very frequently in certain epidemics, and may be attended by intestinal hæmorrhage. Nephritis is a rare complication. Various affections of the nervous system, which are described elsewhere, occur in rare cases.

The **diagnosis** of measles in a well-marked case is easy, owing to the special course of the symptoms and characters of the rash. Occasionally, when the throat symptoms are severe and the eruption more diffuse and less papular than usual, there may be a considerable resemblance to scarlet fever. But the history of the case, the long primary stage, and a careful inspection of the rash will generally prevent error. The prodromal rash of small-pox may resemble closely that of measles in an early stage, but in the latter the papules

which shortly appear are less shotty, and the accompanying symptoms of catarrh differ from those usually observed in small-pox. The diagnosis from rubella, which may be extremely difficult in a single case, is discussed under that disease. Acute dermatitis occasionally causes some hesitation, but the history of the case and a careful inspection of the eruption, which commonly presents pustular points and scabs and runs a more chronic course with less fever, will prevent mistakes.

The **prognosis** depends almost entirely upon the nature and severity of the complications, the large death-rate which measles produces being due in the main to pulmonary complications. High temperature is not necessarily a bad omen; on the contrary, the worst cases are seen in debilitated children who pass into a condition of great depression, attended by hæmorrhage into the rash, without high pyrexia and, it may be, with normal or subnormal temperature. The existence of chronic tuberculosis is an unfavorable element in prognosis, since an attack of measles may determine a rapid extension.

The **treatment** of measles in mild cases does not call for active measures. The child should be kept in bed from the time the temperature rises until four or five days after the pyrexia has ceased. The room should be shaded to diminish the distress caused by the photophobia, but not kept too dark. The food should be light, consisting of diluted milk, vegetable soups, and meal gruels. As a rule, pyrexia does not call for any direct treatment, but, when high, it may be reduced by the wet pack with water at 85° F. The prophylaxis of broncho-pneumonia is, in reality, the most important part of treatment, and in this cleanliness and ventilation are of the first importance. For the constipation, which often exists at the beginning of the disease, mild laxatives only, such as castor oil or liquorice powder, should be given, since purgatives tend to produce the intestinal catarrh or membranous colitis, which constitutes one of the dangers of the disease. Great care should be exercised during convalescence, both in diet and in guarding the child from chill, though it should be given the advantage of outdoor air as early as the weather permits.

Rubella, or German measles, is an acute, specific, infectious, eruptive fever, which resembles a mild attack sometimes of measles, at other times scarlet fever. As a rule, it is a very mild disease, and is, indeed, chiefly of importance because it is liable to lead to mistakes in diagnosis.

The **incubation** period is long, seventeen or eighteen days as a rule; but it may be two or three days more, or five or six days less. The patient is infectious for some days before the appearance of the rash, but not for long after its disappearance. Infection is over

within three weeks after the beginning of the attack. The time for which a susceptible person, who has been exposed to infection, must be kept under *observation* before it can be asserted that he has escaped the disease is three weeks, and at the end of this time he must be found free from rash, sore throat, or glandular enlargement.

Often the first symptom to attract attention is the rash. In other cases its appearance is preceded for from twelve hours to two or three days by malaise, headache, suffusion of the eyes, soreness of the throat, pain in the back, and glandular enlargement. The rash is seldom delayed beyond the second day. It appears first behind the ears and round the mouth and nose; it spreads thence, often very rapidly, but sometimes in successive crops, to the trunk and limbs. At first it consists of slightly raised, rosy-red spots, scattered over the healthy skin; the spots are smaller and more discrete than those of measles, larger and more papular than those of scarlatina. In some cases—those which constitute the scarlatiniform class—the character of the rash changes after a few hours. On the face it is obscured by a bright red flush, while the limbs become covered by a fine, punctate rash, identical with that of scarlatina. The rash, whether morbilliform or scarlatiniform, reaches its maximum in any area in about twelve hours, and then begins rapidly to fade. It is all gone by the third day, as a rule. [Coincidentally with the appearance of the exanthem is observed the enanthem, different, however, from that of measles, described above. According to Forchheimer this enanthem is the same eruption that is found on the skin, is of a pure pinky rose red, and is localized upon the uvula and soft palate, rarely invading the hard palate. It is short-lived, fading away within the first twenty-four hours.] The general symptoms, which are usually very mild, are coincident with the rash. The temperature rises, as the rash comes out, to perhaps 100° or 101° F. It remains at about this level, or perhaps touches 102° F. on the evening of one or two days while the rash is out, and falls to normal as the rash fades. The pulse increases in frequency as the temperature rises, and decreases as it declines. The eyes are red and watery, but, usually, there is no photophobia; there is a general redness of the palate and fauces, and often enlargement of the tonsils and some dysphagia. Cough, which is often troublesome, is generally dry and ineffectual, and only occasionally are there signs of bronchitis. The patient, as a rule, does not feel ill, and when first seen will often be found running about, retaining his appetite and taking only a curious interest in his rash. The most characteristic symptom is a general enlargement of the glands; this may precede by three or four days the appearance of the rash, and may continue for a week or more after it has faded. The glands enlarged are those at the back of the neck, beneath the ear, and under the sterno-

mastoid muscles ; more rarely, those in the axillæ and groin. They are hard and tender, though, as a rule, the patient makes no complaint of them. Convalescence begins as the rash fades, and is generally rapid. Desquamation occurs in a large proportion of the cases ; it is often scanty, and to be detected only by careful examination of parts of the body, such as the sub-clavicular regions, which are not much exposed to friction. It is commonly more copious in those cases in which the rash most resembles that of scarlet fever, but it is always fine and branny, and even on the hands and feet the epithelium is not detached in flakes. Complications rarely arise. The throat affection may be severe, and a friable or pultaceous false membrane has been known to occur on the tonsils. The bronchtis which sometimes accompanies the rash may be severe, and may then persist after the rash fades. Broncho-pneumonia and pleuro-pneumonia have been known to supervene. Laryngitis may occur, but is seldom or never serious. The pharyngeal catarrh may cause obstruction of the Eustachian tube and pain in the ear. Relapse after an interval of a few days or two or three weeks has been observed.

The **prognosis** is good ; in cachectic children already suffering from some chronic wasting disorder, an attack of rubella may hasten or determine a fatal issue, or may leave chronic tonsillitis or naso-pharyngeal catarrh or chronic adenitis. As a rule, recovery is rapid and complete.

Aberrant types are described. It is probable that the disease may occur without rash. In some cases suffusion of the conjunctiva, "pink eye," with slight feverishness, may be the only symptom, and probably some of the cases of widespread enlargement of the lymphatic glands, with slight feverishness, in children are examples of rubella without the rash. Epidemics of a *rose rash*, a papular eruption without catarrh and with little or no fever, occur sometimes in summer, and are generally classed as mild rubella. They are probably due to some different infection, which does not protect from rubella. This rose rash consists of large rounded areas of bright red, closely set spots, scarcely raised, which appear suddenly without prodromal symptoms on the neck, limbs, and trunk. The face often escapes. The rash fades in about thirty-six hours, and the pyrexia (if there has been any) ceases with it. The fauces may be a little reddened, but there is no complaint of sore throat and no enlargement of glands.

The **treatment** of an ordinary case of rubella should consist merely of keeping the child in bed or in a warm room for four or five days, and indoors for three days more. It should then be given as much fresh, outdoor air as possible for a week, and a series of disinfecting baths. The diet should be light during the pyrexia, and it is well to give a dose of laxative medicine at the onset of the disease.

The **diagnosis** from measles or from scarlet fever may often be—at any rate at first—extremely difficult, if not impossible. The Medical Officers of Schools Association admit that "In some cases the eruption may so closely resemble that of either measles or scarlatina in local appearance that a diagnosis founded on the eruption alone is impossible." Probably the most distinctive feature is the early and general glandular enlargement. The mild type of the catarrh, the absence of photophobia, and the change in the character of the eruption when this occurs, will assist in the discrimination from measles; while the absence of the thick white fur on the tongue, which peels off from the tip and edges on the fourth day, leaving a raw, red surface, the general absence of albuminuria, and the duskier red of the eruption, will help to distinguish the cases from scarlet fever; at a later stage the character of the desquamation will give valuable evidence. Children seldom show much change of temper with rubella, whereas they are generally irritable and depressed with measles, and feel very ill with scarlet fever, except in the mildest attacks. In arriving at a decision, all the circumstances of the case must be taken into consideration, but too much weight must not be attached to a previous history of measles or scarlet fever, especially if the diagnosis rests merely on maternal authority. The table on the next page contains a list of the points to which attention may specially be directed.

In **scarlet fever** the incubation period is short—usually from twenty-four to seventy-two hours. It may be shorter, or may extend to four, five, or seven days. Scarlet fever is infectious from the onset of the earliest symptoms and until long after convalescence has been established. Infection may persist certainly as long as desquamation, so that it may still be active eight weeks after the onset. Infection is readily preserved and conveyed by fomites. A susceptible child who has been exposed to the infection must not be assumed to have escaped unless on the eighth day he is free from fever and sore throat.

The *onset* is rapid, often extremely sudden. It is attended commonly by vomiting, in young children by convulsions, and the temperature is then found to be elevated. The skin is red, pungently hot to the touch, the tongue is furred, and the throat dry. The *rash* appears usually within twenty-four hours of the first symptoms. At first a streaky redness of the neck and chest, upon which are situated closely scattered red points, it spreads rapidly, and when fully developed the whole surface of the body is of a vivid scarlet tint, though occasionally areas of normal color may remain. The face is usually spared, and the rash, if present, is confined to the forehead and temples, the cheeks being merely flushed. Sudamina frequently occur, and may be very numerous, and occasionally there are petechiæ.

	Rubella.	Measles.	Scarlet Fever.
Incubation period.	9 to 21 days; usually 18.	5 to 14 days; usually 10.	Usually about 2 days.
Prodromal symptoms.	Short and slight. Little or no depression.	3 to 4 days; generally marked. Depression generally marked; often much prostration.	Usually brief. Vomiting frequent. With much rash much depression.
General symptoms.	Tongue clean or slight fur; appetite often retained. T. may be normal. Pulse little altered, or accelerated in proportion to fever.	Tongue furred; little or no appetite. T. usually 100° or more. Pulse generally accelerated in proportion to fever; often weak and dicrotic.	Tongue coated; peeling on 4th day producing "strawberry" T. T. always raised, often 105° to 106°. Pulse always accelerated; commonly out of proportion to fever.
Rash.....	1st or 2d day, commonly first symptom; rosy-red dots. First, or early, about mouth. Always patchy.	4th day. Papular, brick-red and crescentic; appear about mouth or forehead.	Diffuse, dusky red. Face usually escapes.
Catarrhal symptoms.	Redness of throat, diffuse. Conjunctivæ suffused; slight lachrymation. Bronchitis slight; broncho-pneumonia rare. Diarrhœa absent.	Redness, patchy at first. Catarrhal conjunctivitis. Much lachrymation and photophobia. Bronchitis usually marked; broncho-pneumonia common. Diarrhœa frequent.	Proportionate to skin eruption. Dusky red. White plugs in tonsillar glands. Conjunctivæ unaffected. Lung complications uncommon. Diarrhœa absent.
Lymphatic glands.	Enlarged, tender and hard; including posterior cervical, axillary and inguinal.	Enlargement generally late and limited to those about the angle of the jaws.	Enlargement of cervical proportionate to faucial affection.
Albuminuria.....	Rare and slight.	Very rare.	Frequent.
Convalescence	Rapid.	Commonly more protracted.	Often prolonged owing to complications.
Desquamation.....	May be copious; always fine.	Seldom copious; always fine.	Generally copious; in shreds.

After about two days the rash commences to fade. The tongue is at first furred in the centre and red at the tip and edges, the enlarged papillæ showing through the fur as red points (strawberry tongue). As the rash subsides the tongue cleans, leaving a red, rough surface (raspberry tongue). The pharyngeal symptoms vary greatly. In some cases there is merely a red mottling, which appears about the same time as the rash. In others, one of the earliest symptoms is severe follicular tonsillitis, which may lead to sloughing of the tonsils. In others, again, the inflammation is more diffuse, affecting all the pharyngeal structures, and leading to secondary adenitis and to ulceration, which may be so deep as to open into the carotid. The adenitis at this stage is proportionate to the extent and degree of the local lesion.

The course of the *temperature* in a typical case is as follows:— After the sudden rise at the onset there is a slight remission, followed by a second rise, reaching a maximum of 103° to 105° F. on the second or third day; thereafter there are morning remissions and evening exacerbations for four or five days, the normal temperature being reached on the eighth or ninth day. Febrile albuminuria is frequent, but disappears as the temperature declines. After the disappearance of the rash the skin becomes harsh and dry, and desquamation commences usually on the forehead and neck. The epidermis is detached in large scales, which upon the fingers may be so large as to resemble the fingers of a glove. The hair falls out, and may even be lost entirely. Desquamation is usually over in from ten to twenty days, but may last much longer, and a second and even third desquamation may occur.

The severity of scarlet fever varies very greatly. In some cases it is extremely mild, and the diagnosis can only be made owing to the simultaneous occurrence of other cases, or the subsequent occurrence of cases infected from the mild case. In others the disease sets in with delirium, headache, high fever, and even hyperpyrexia. Great prostration rapidly ensues, with dyspnœa and a rapid feeble pulse, and the patient becomes comatose. Occasionally, especially in debilitated children, the rash is accompanied by petechiæ and hæmorrhage from the nose or kidneys; prostration is great, and the result is usually fatal.

At an early stage the most frequent *complication* is otitis media, which arises in connection with the throat lesions. Membranous pharyngitis and laryngitis are observed in a considerable proportion of the more severe cases, but as a rule this membrane is really diphtherial. Scarlatinal *nephritis* is the most characteristic sequela. It begins usually in the second or third week, but may be delayed until the fourth or fifth. It varies very greatly in severity, and may come on after a mild attack. It is discussed elsewhere. Occasion-

ally œdema without albuminuria, due in most cases probably to anæmia, is observed. Both pericarditis and endocarditis may occur, but the latter is often unrecognized during the fever, and only gives rise to symptoms some months later. Arthritis affecting sometimes many joints is an occasional complication, observed usually as the fever subsides, but occasionally at an early date contemporaneous with the initial tonsillitis. It is in some cases associated with pericarditis; recovery without permanent injury to the joints is the rule. Arthritis commencing at a late date, and limited to one or two large joints, is more apt to end in suppuration. Mahomed believed that the interval from the eighteenth to the twenty-second day was a kind of critical period during which there was a rise of arterial tension, diminished excretion of urine and urea, and rise of temperature, with a corresponding liability to complications—albuminuria, otorrhœa, diarrhœa, and cervical adenitis quite out of proportion to any local pharyngeal lesion remaining. At about the same period secondary rashes are met with in 2 or 3 per cent. of all cases, but rather more frequently in children under five years (4.8 per cent.).[1] Erythematous, urticarial, and purpuric rashes are most often seen in cases complicated by arthritis; papular and eczematous rashes are also met with at about this period, and are probably, in many instances, due to septicæmia.

Pulmonary complications are, on the whole, uncommon, but in some epidemics broncho-pneumonia occurs in a large proportion of the cases, and occasionally pleurisy, which is usually purulent. Of the nervous complications the most common is chorea, which is seen most often as a sequel to those cases in which arthritis and heart disease have occurred.

The diagnosis of scarlet fever is usually easy. The errors which occur are rather in the direction of mistaking other conditions for it than of overlooking the disease itself. Acute exfoliative dermatitis, which may come on suddenly, with fever and a rapidly-spreading erythematous rash, cannot always be distinguished with certainty in the earliest stage from scarlet fever; but even when doubt exists, owing to the absence of throat symptoms, it is prudent to treat the case as though it were certainly scarlet fever. The cases classed as erythema scarlatiniforme are discussed elsewhere. The resemblance of rubella in one of its forms to scarlet fever is sometimes very close, as is mentioned elsewhere. The rash produced by septicæmia may be identical with that of scarlet fever. The throat symptoms are usually absent or slight, but as operation or injury predisposes a child to contract scarlet fever, the management of cases in which any doubt arises should be founded upon the assumption that the disease may be scarlet fever.

[1] Manning, *Lancet*, 1892, ii., p. 363; *Conf.* Caiger, *ibid.*, 1891, i., 1249; and Symes, *Bristol Med. Chi. Journ.*, March, 1897.

Membranous laryngitis, coming on after the third or fourth day, is usually due to diphtheria, but earlier than this, especially if the membrane is thin and white, it is probably due to streptococcus-infection. The rash produced by belladonna has been mistaken for that of scarlet fever, as have also those produced, more rarely, by quinine and potassium iodide. The absence of fever, the condition of the throat—which in belladonna poisoning is red and dry, but not inflamed—and the general circumstances of the case will prevent mistake being made.

There is considerable difference in **prognosis** in different epidemics. Unfavorable symptoms are: fever which is very high or continuous, severe nervous symptoms, hæmorrhages, or extensive angina with adenitis. The mortality is higher the younger the child. A moderate amount of albumen in the urine in an early stage is not a serious symptom, and its danger in convalescence is in proportion to the rapidity with which it develops.

The **treatment** in mild cases consists mainly in attending to the comfort of the patient and warding off complications. The child should be isolated in a well-ventilated room, and should be given a limited light diet, consisting of diluted milk and gruels, and allowed to drink freely of water, which may be acidulated. Sponging with warm water two or three times a day, and, when desquamation begins, a warm bath with superfatted soap should be given daily. Very good results are claimed[1] for the systematic use of warm baths, twice a day for the first week, and then daily. Each bath lasts ten minutes. The liability to nephritis, it is maintained, is thus diminished, owing to the baths favoring the removal of the poison which is assumed to be eliminated by the skin. The course of the disease is rendered milder, and desquamation during convalescence is slight, owing to the gradual removal of the desquamating epidermis by the baths. The use of oils and ointments for the skin is of doubtful advantage to the patient, and, when isolation can be effectually carried out, is unnecessary in the interests of others. Very high temperature, accompanied by delirium or collapse, should be treated by cold sponging, or by a cold or lukewarm pack. When, on the second or third day, the temperature is found to be rising rapidly, a warm bath, cooled to 80° or 85° F., will check the rise and relieve the attendant symptoms. It may be combined with or replaced by the use of the ice-cap. The throat symptoms will be relieved by cold drinks or small pieces of ice to suck, and by cold compresses or hot fomentations externally. Local astringent applications, such as glycerine of tannin or pulverizations of resorcin, should be used also, and, when ulceration has commenced, insufflation of boric acid in powder, and the local application of a strong solution of nitrate of silver or of

[1] Schill, *Jahrb. f. Kinderhlkde*, Bd. xliii., s. 260.

chloride of zinc to the part are to be advised. The occurrence of secondary rash at or about the end of the third week should be taken as an indication that the onset of other complications is not improbable. In the hope of preventing nephritis, or diminishing its severity, the patient should be sent back to bed, placed on a very bland diet (milk), and given a dose of calomel followed by acid tartrate of potash.

[**Bacteriology.**—Class of Chicago believes he has discovered the specific organism of scarlet fever. He describes it as follows: a diplococcus resembling a very large gonococcus, growing best on a medium made of glycerin-agar and garden earth, and stained by a number of the aniline dyes.

Pearce has studied the bacteriology of many complications of scarlet fever and states that the micro-organisms producing secondary inflammatory lesions are the streptococcus pyogenes, the staphylococcus pyogenes aureus and the pneumococcus. He considers that infections of the middle ear, antra of Highmore and of the sphenoidal sinuses are of the utmost practical importance and apt to be the starting point of chronic trouble.]

CHAPTER VII.

ACUTE SPECIFIC INFECTIOUS DISEASES (*continued*).

Influenza—Whooping-cough—Mumps—Glandular Fever—Cerebro-spinal Meningitis—Enteric Fever.

Influenza, an acute infectious disease spread almost solely by personal intercourse, and due, in all probability, to the specified bacillus described by Pfeiffer and Kitisato, affects children in many epidemics in about the same proportion as adults. Infants at the breast enjoy a certain immunity, but it is less marked than in many other diseases of this class. In England and Wales in 1890 the deaths from influenza per 1,000 living at different ages were:[1] for all ages, males 0.17, females 0.14; at ages 0–5 years, males 0.16, females 0.12; at ages 5–10 and 10–15, males 0.02, females 0.03.

The various *clinical types* described as occurring in adults may also be observed in children, but in them a large proportion of all cases in most epidemics are of the simple *febrile type;* that is to say, pyrexia without any definite signs or symptoms referable to any one organ or system. The onset of the fever is usually very sudden, and the thermometer often attains 105° to 106° F. in a few hours; the skin is flushed and moist, and the child is usually drowsy. Occasionally somnolence is very marked, so that the child cannot be aroused. In those cases in which the temperature rises to the height mentioned defervescence occurs usually in about twenty-four hours or even less, and after a few days of languor the health is completely restored. In other cases, especially those in which the onset is less sudden and the initial rise of temperature less high, the pyrexia continues for two, three, or four days, and convalescence is more prolonged. Allied to this type are those cases in which *nervous* symptoms are marked; the patient, if an infant, is restless and cries out occasionally, and in older children complaint is made of neuralgic pains, sometimes referred to the joints and then called rheumatic. In infants convulsions sometimes occur; in older children vomiting; and at both ages somnolence is occasionally very marked, and causes much alarm to the friends. The *catarrhal* type is also observed with great frequency. The catarrh affects all the respiratory passages and the

[1] H. Franklin Parsons, "Further Report (to the Local Government Board) and papers on Epidemic Influenza," 1889–92.

conjunctivæ, so that the aspect of the patient recalls the onset of measles. In other cases the buccal mucous membrane is that most severely affected, and small circular ulcerations are often observed. In other cases, again, the general symptoms—flushed face, moving alæ nasi, and rapid breathing—suggest pneumonia, but the physical signs are those of slight bronchitis only, and the dyspnœa is probably due, in the main, to toxæmia. Occasionally the signs of laryngitis are marked, the voice is hoarse, and attacks of stridulous breathing occur, during which there is marked recession of the bases of the chest. These symptoms may persist without much change for several days, and then disappear rapidly. Convalescence from the catarrhal form is usually more prolonged than from the simple febrile form, and frequently definite bronchitis develops, and sometimes broncho-pneumonia. The *gastro-intestinal* type is perhaps less common in infancy and childhood than might have been expected, judging from the liability of children to such affections. Soon after the onset of the fever the child begins to vomit, and the tongue is seen to be covered with a thick white fur, or is red and irritable, with enlarged papillæ. Diarrhœa, the stools containing usually much mucus, comes on, and the patient loses flesh rapidly. After three or four days the diarrhœa tends to diminish, but some looseness of the bowels often persists for several weeks, and the strength and flesh are regained slowly. In some cases belonging to this type the symptoms are very severe; the temperature is high; the child cries with pain in the belly, and is drowsy or somnolent; the hands and tongue are tremulous; and the whole aspect recalls typhoid fever. In some there is diarrhœa, in others constipation. In others, again, the typhoidal condition becomes established more slowly after an ordinary milk attack of the gastro-intestinal form. In cases having this character the symptoms may persist for a fortnight or more.

The most common and serious **Complication** is broncho-pneumonia. It occurs most often in cases of the catarrhal type but may complicate any form. It may present no symptoms which in any way distinguish it from ordinary, so-called primary, broncho-pneumonia; or the temperature, dyspnœa, and general distress may be out of proportion to the physical signs at any time to be detected; or it may from the first be of a peculiar depressed type, which has been well described by Ferreira.[1] In these most characteristic forms the temperature is little if at all raised, cough is not troublesome, and the patient, usually a child under two years of age, is listless and drowsy. Dyspnœa is severe, the face dusky, the respiration hurried, with recession of the chest walls in their lower part and under the clavicles. The whole course of the case is prolonged and the symptoms throughout of asthenic type. Pulmonary collapse is very apt to occur and to be

[1] *Rev. d. Mal. de l' Enf.*, 1895, p. 105.

the determining cause of death, which is the termination of a large proportion of such cases. Pneumonia, lobar in distribution, also occurs as a complication, but less often than in the adult. Pleuro-pneumonia is a not uncommon complication in some epidemics, and may be followed by empyema. [**Otitis Media** is also a frequent complication of influenza. Nephritis occurs not uncommonly and repeated examinations of the urine should be made.]

The **diagnosis** of influenza, unless an epidemic is known to exist, is often very difficult. [It may often be determined by bacteriological examination of cultures taken from the mouth and throat showing Pfeiffer's bacillus.] During epidemic periods the febrile form is sufficiently characteristic, especially if accompanied by somnolence. The resemblance of the severer types of the gastro-intestinal form to typhoid is considerable, but the exanthem is absent.[1] The flushing which commonly attends the onset of influenza may be so intense as to amount to erythema and later on may be followed by desquamation, so that a considerable resemblance to scarlet fever is presented. In other cases the erythema is patchy and presents some similarity to that of measles. The diagnosis of such cases must depend partly on a careful examination of the rash, which resembles, but is not identical with, that of scarlet fever, and on the general circumstances of the case. Tonsillitis is sometimes severe, and may increase greatly the difficulty of diagnosis, but such a combination is rare. The resemblance to the rash of measles is generally very superficial, and the date of appearance of the rash does not coincide with the epoch at which it appears in measles.

The **prognosis** in children over three or four years of age is very good, the main danger being from the severity of the broncho-pneumonia when it complicates the malady. In younger children the prognosis is also good, except when the patient is already rickety or "scrofulous"; in the one, broncho-pneumonia, if it occur, is a serious disorder, as is always the case in rickety children, and has a prognosis of its own; in the other, the inflammatory affections of the air passages, which attend influenza and are always associated with more or less enlargement of the lymphatic glands, may determine severe adenitis, and even light up acute tuberculosis. In infants who are, comparatively, seldom affected, influenza is often severe, the nervous depression being very marked and the mortality higher than at other ages of childhood. After an attack of influenza, at any age, which has been complicated by broncho-pneumonia or much gastro-intestinal disturbance, convalescence is often prolonged, and the child suffers from anæmia, loss of appetite, and languor. Tuberculosis is an occasional sequel.

[1] The diazo-reaction is not obtainable. The typhoid serum reaction will also, doubtless, be of use in the future.

Prophylaxis is not easy. The incubation period is two or three days, as a rule, and a patient begins to be infectious certainly within the first twenty-four hours, and so continues for eight or ten days, probably therefore after he has become sufficiently convalescent to resume his ordinary avocations. Children residing in the country and much out of doors are more likely to escape, or if they contract the disease to suffer from a milder attack, than those in towns and under unfavorable hygienic conditions (especially overcrowding). The first member to be attacked in a household is commonly an adult, usually the father, and immediate removal of the children often serves to save them from the infection. An infant at the breast may escape, even though the mother suffers. When a child has been attacked, attention to the cleanliness of the mouth and pharynx, and to the ventilation of the room in which it is nursed, will diminish the risk of pulmonary complications.

The **treatment** of influenza should be as simple as possible. The patient should be kept in bed, given liquid nourishment and demulcent drinks to appease the thirst, which is often troublesome. Depressing antipyretic drugs should be avoided, as the danger in almost all cases lies not in the fever but in the accompanying or sequent nervous depression. Quinine, which has been much used, especially in the form of the ammoniated tincture, is at least harmless, and sodium salicylate is of some value, especially in cases in which there are neuralgic or arthritic pains. Broncho-pneumonia and diarrhœa must be treated on ordinary principles. Restlessness and excitement may be treated by phenacetin, of which as much as gr. j may be given every four hours at one year of age. The warm pack, if the temperature be moderate, or the cold pack if it be very high, has also a very soothing effect. The most important part of treatment in the great majority of cases is the management of convalescence. The patient should be kept in bed for at least three days, even if the temperature has fallen early, and should not be allowed out, if the weather be cold or damp, for another week. After this, exercise in the open air should be taken with proper precautions. In school children, especially those of neurotic type, a rest of several weeks should be advised, and a fortnight after the onset a change to a bracing climate can usually be borne. In cases, however, in which there has been protracted broncho-pneumonia much care should be exercised for months, and if the child fail to regain strength and weight in a satisfactory manner, it will be well that the cold and wet months should be spent, if possible, in a warm and dry climate. This applies especially to those having a tuberculous family history, and to those who have already presented tuberculous manifestations (adenitis, etc.).

Whooping-Cough (pertussis) occurs in epidemics, and is the source of a very large mortality among children. It is most preva-

lent and serious during the period of its first dentition, but may occur in infancy. It is comparatively rare after the age of ten years. It is extremely infectious in the prodromal stage, when the symptoms are not characteristic, and the infection may be carried by fomites. A patient becomes infectious as soon as the catarrh sets in, and before the characteristic whoop develops.

The infective agent has not been certainly identified. The mucus expectorated at the end of a paroxysm contains small yellowish lenticular masses containing a small diplococcus, which, according to Ritter and others, is not found under other conditions. This organism appears to be confined to the lower part of the trachea. The morbid anatomy of whooping-cough is in the main that of its complications—bronchitis and broncho-pneumonia in particular. The only lesion at all characteristic is slight tracheitis, the reddened and swollen mucous membrane being covered by a very tenacious mucus.

The *incubation period* varies a good deal, probably from five to thirteen days. It is succeeded by a *catarrhal stage*, during which the child suffers from coryza, which becomes complicated by more or less severe bronchitis, and often by broncho-pneumonia. The cough becomes worse, and by degrees paroxysmal, until at the end of a week or ten days the *paroxysmal stage* becomes established. When well developed, a paroxysm is extremely distressing to witness. The child's face assumes an expression of painful apprehension; cough, which it tries to suppress by holding its breath, then begins; the coughs succeed each other more and more rapidly, until the chest is in extreme expiration. The face is red or purple, the eyes suffused, a thin mucus runs from the mouth, and the child stands clutching a chair or its nurse's apron. After a short pause, during which the chest is motionless and no sound is uttered, a long inspiration is taken which is accompanied by the characteristic whoop, a long-drawn, high-pitched full note, to be heard for a great distance. The whoop may be repeated two or three times with diminishing intensity, or the first inspiration may be imperfect and the whoop slight, the succeeding whoop being very loud. The paroxysm may now end, or the cough may begin again, ending again in a whoop. Finally, after several such attacks the paroxysm ends with the expulsion of a thick, tenacious, but usually clear mucus. This is effected by a kind of pharyngeal regurgitation, which is not true expectoration, and may be observed in infants. Very often the regurgitation determines true vomiting, any food in the stomach being expelled along with the mucus. When the cough has existed for some time the muscles of the upper part of the abdomen become very tender, and every movement of them is painful. In the latter stages of the disease the paroxysms are often more dreaded on account of the pain thus produced than for their severity. Hæmorrhages from the

mucous membranes of the nose and throat are not uncommon during the paroxysmal stage, and subconjunctival ecchymoses are very often produced. They are apparently due to the extreme venous congestion caused by the expiratory spasm which precedes the whoop. After a paroxysm, or a series of paroxysms, the child is often left in a condition of great exhaustion, amounting sometimes to collapse, which lasts for half an hour or more, during which time it lies motionless, limp and apparently unable to move. In some cases loss of sensation has been associated with the paresis. In others, various forms of sensory aphasia have developed and have persisted for several weeks. Sometimes the child is able to speak, answer questions, and recognizes people and objects, but is unable to find the names of objects with which it had been perfectly familiar. In other cases, again, the power of speech is almost completely lost, only a few simple words being retained, and the patient is unable to recognize persons and objects with which it was well acquainted (apraxia). In some of the cases in which nervous symptoms of this order occur, general convulsions take the place of the exhaustion following the paroxysms ; in others, the spasmodic movements are limited, as in one case recorded by Troitzky, in which there were facial convulsions, irregular movements of the eyes, and retraction of the head. The convulsions, however, may be followed by paralysis, which has usually a hemiplegic distribution. The paralysis may be limited to the muscles supplied by one or more cranial nerves, as in the case shown in the illustration on the opposite page, in which the sixth and seventh nerves on the left side were paralyzed. Or it may be a regular hemiplegia affecting the face, arm, and leg on one side, and has been proved to be due to hæmorrhage in some cases, but in others recovery takes place in a few weeks, and is so complete that it is difficult to suppose that the symptoms can have been due to actual extravasation.

The paroxysmal stage endures in mild cases two or three weeks only, but the average is perhaps five weeks. Very often, after the paroxysms have diminished in number to two or three in the twenty-four hours they again become frequent, owing in some cases to an attack of coryza. After the characteristic whooping attacks have ceased, the child usually suffers for some weeks, often for months, from paroxysmal cough with slight chronic bronchitis. This is particularly the case in the winter and spring seasons, during which cough and bronchitis are apt to persist until the weather becomes genial. Many cases of chronic winter bronchitis in children date from an attack of whooping-cough. The most important *complication* is broncho-pneumonia (*q. v.*), which is the cause of the great mortality attributable to whooping-cough, and is seldom absent in fatal cases. Broncho-pneumonia comes on usually during the early part

of the paroxysmal stage, and its onset is attended by sudden rise of temperature dyspnœa, and usually by cessation of the paroxysmal cough and whoop. Tracheitis and bronchitis are present to some extent in most, if not in all, cases of whooping-cough. The bronchitis is sometimes very extensive, and is then a serious menace to life, not directly so much as by the extra strain it puts upon the heart already

FIG. 8.

Paralysis of the Sixth and Seventh Cranial nerves coming on during Whooping-Cough, and due probably to limited hæmorrhage into the Pons (Dr. Craig's case, *Brit. Med. Journ.*, 1896, vol. i., p. 1440).

strained by the congestion of the right side produced by the paroxysms. In such cases the dyspnœa may be extreme, the face livid or purple and swollen. In slight cases there is pallor and œdema of the lower eyelids.

The **diagnosis** of a well-marked case of whooping-cough during the paroxysmal stage is easy, since the paroxysm of coughing ending in the high-pitched whoop is characteristic. The only condition which

resembles it nearly is that paroxysmal cough and inspiratory stridor produced by enlargement of the tracheo-bronchial glands (*q. v.*). During the catarrhal stage, however, diagnosis may be impossible, unless the child is known to have been exposed to infection. If there be much broncho-pneumonia the paroxysms and whooping usually do not appear, or are suspended until it begins to resolve. The existence of ulceration of the frænum linguæ may increase a suspicion already existing, but is not in itself characteristic, especially in young children who have recently cut the lower incisors. The mistakes most liable to be made are to overlook whooping-cough in the early stage, or to attribute the paroxysmal cough of enlarged glands to a mild attack of whooping-cough.

The treatment is unsatisfactory because no remedy has much effect on the duration of the malady, and because it is impossible to foresee which one of the numerous drugs at our disposal will have the best effect. The main indications which we can hope to fulfill are to diminish the number and severity of the attacks and to prevent complications. Expectorants are valuable in the early stage, especially ipecacuanha with which antipyrin may usually be combined with advantage; the dose of the latter drug should be at first a grain for each year of life three times a day. Belladonna, which is much used, is often very effectual in lessening the severity of the paroxysms. It is the best drug for infants and young children, but must be given in sufficient doses (ext. gr. $\frac{1}{8}$, tinct. ♏ iij–ij) to an infant three or four times a day. The production of dryness of the throat should be avoided, but slight flushing of the face should follow each dose (Jacobi). Bromoform in some cases diminishes the severity of the paroxysms after a few days, but it is not suitable for young children, and is uncertain in its action, as is also cocaine, which has been recommended in doses of gr. $\frac{1}{16}$ for an infant, gr. $\frac{1}{3}$ for a child of six years, thrice a day. [Bromoform may be given as follows:

Bromoform,	♏ij
Ol. Amyg.,	♏x
Trucil Tragacant.,	♏xv
Aq. Caru.,	ad ℥j

Appendix.]

In older children good results are sometimes obtained by giving quinine (sulphate or hydrochlorate gr. iij t. d.) or the tannate, which is less bitter, in powder (gr. vj–x t. d.). Oxymel of squills, ʒiij–iv, in divided doses during the afternoon for a child of five has been recommended, and the drug in some cases has a beneficial action. When there is œdema of the face, and weakness of the heart, it is well to give small doses of digitalis, and to increase them gradually if necessary. When sleep is much disturbed by the paroxysms two or

three doses of potassium bromide taken during the afternoon and evening often procure a quiet night.

Local treatment directed to the upper air passages is not to be neglected. The nose should be kept clean, and a small quantity of borac acid ointment (to which menthol, gr. xx to ℥j may be added) should be introduced into the nose two or three times a day. Nasal insufflations have also been strongly recommended; for this purpose powdered benzoin and lycopodium equal parts, or bismuth salicylate five parts, benzoin five parts, quinine sulphate one part, may be used. Local applications of antiseptic solutions to the pharynx and upper orifice of the larynx undoubtedly do good; indeed I have seen more striking results from the application of a solution of recorcin (2 per cent.), as recommended by Moncorvo, than from any other method of treatment, but like all other remedies, it fails more often than it succeeds. The diffusion of terebinthinate vapors through the room, as by the old fashioned method of stirring Stockholm tar with a hot poker, gives relief, and advantage is to be derived from diffusing turpentine through the air by evaporation.[1]

"Hygienic treatment" is of the first importance. The child's bedroom should be well ventilated by night, and thoroughly aired by day. It should spend as much time as possible out of doors every day, and as soon as convalescence is established change of air is to be recommended, by preference to a dry elevated site.

The diet through the height of the attack should be light and nourishing. When vomiting is very troublesome sedatives may become necessary—morphine, codeine, or cocaine—but their use may often be avoided by giving liquid food only, either iced, or as hot as can be taken, and choosing the period shortly after a paroxysm for its ingestion, the child being made to lie down for a short time. Most important is the prophylaxis of broncho-pneumonia. The risk of this complication, possibly also the severity of the disease itself, is increased when many children are treated together in the same room or ward, and under such circumstances the most rigid precautions should be observed as indicated in the chapter on broncho-pneumonia.

[Pertussis, like measles, acts as a predisposing cause of tuberculosis by diminishing the general resisting power of the patient and thus rendering the mucous membrane of the respiratory tract susceptible to invasion by the bacillus of tuberculosis. Hence it is important to watch carefully debilitated children, and those with a tuberculous heredity for several months after apparent convalescence and to secure for them the best hygienic and dietary conditions pos-

[1] It has been recommended to burn sulphur in the bedroom about five hours before bedtime and to keep the room closed until just before the child is put to bed. Ullmann burns sulphur thrice a day for a quarter of an hour in the room in which the child is.

sible. The practice commonly in vogue of allowing patients with pertussis to return to an out-patient clinic is criminal and cannot be too severely condemned.]

Mumps (*Epidemic Parotitis*) is an acute infectious disease characterized by inflammation of the salivary glands, usually the parotid.

The disease affects children (five to fifteen years) mainly, and both extremes of life are almost immune. It is disseminated mainly by personal communication, and is extremely infectious, especially in the early stage. The infectiousness of a patient diminishes progressively from the time of onset of the parotitis, and has ceased in at most three weeks from that time.

The *incubation* period, *i. e.*, the interval between exposure to infection and the onset of parotitis, is usually three weeks, but may be a few days longer, or as much as a week shorter.

The *prodromal* period lasts three or four days, during which the patient is capable of transmitting the infection, but the symptoms are slight and not characteristic—malaise, headache, loss of appetite, and sometimes elevation of temperature. With the *onset* of parotitis the temperature rises to 101–103° F., and complaint is made of pain behind the jaw, and difficulty in opening the mouth. Swelling in the parotid region is noticed a little later, usually at first on one side only. In the course of thirty-six to forty-eight hours it becomes very considerable, and has generally begun on the opposite side also. It extends in front of the ear and under the sternomastoid muscle, producing a characteristic deformity. The skin is tense and full, but is not, as a rule, reddened; not infrequently there is extensive œdema of the face and neck. During the enlargement of the gland it is tender and the pain in it may be acute; even if slight it is elicited by any movement of the jaws; for this reason and on account of the mechanical obstruction, and sometimes of spasm of the masseters, there is difficulty in feeding, and the patient speaks "through his teeth." Pharyngitis is present in many cases, and is sometimes accompanied by tonsillitis, but, owing to the difficulty of opening the mouth, its existence can only be surmised in most well-marked cases. Stomatitis is an occasional complication. The parotid swelling begins to subside on the sixth or seventh day, but before this the fever has usually disappeared, and convalescence is rapid in uncomplicated cases. Occasionally the lymphatic glands behind the angle of the jaw are found to be enlarged after the parotid has subsided, and so remain for some weeks. Mumps is usually a mild disease, but sometimes the fever is very high and is accompanied by delirium and prostration. In such cases meningitis has been found, and in most of the comparatively few fatal cases on record has been the cause of death. The delirium is occasionally maniacal, and has been followed by insanity. The other salivary glands, the submaxillary

more rarely the sublingual, are sometimes involved, very rarely the lachrymal gland, still less often the thyroid. The testicles and ovaries are in some cases the seat of an inflammatory affection analogous to that which affects the parotid. Orchitis may precede or accompany the parotitis, and has been known to occur alone (orchitis parotidea). It is very rare in young children, but becomes more frequent after thirteen. The onset of this complication is marked by a rise of temperature, severe pain in the testicle and groin, accompanied often by delirium. It is sometimes attended by purulent discharge from the urethra, and may be followed by atrophy of the testicle. As a rule one side only is attacked. Orchitis occurs in about one-fourth of the cases, and atrophy in about half the cases of orchitis. Ovaritis is much less common. Mastitis may occur in either sex. Vulvo-vaginitis may accompany the ovaritis or occur independently. Suppuration of the parotid is very rare, but even gangrene has occurred. Mumps is often attended by pain in the ear, and by deafness which passes away usually as the swelling of the parotid subsides, but may persist longer, and even be permanent. Laryngitis is a rare complication, but has caused death by œdema glottidis; broncho-pneumonia occasionally develops with great rapidity. Albuminuria is present during the height of the fever in about 30 per cent. of the cases, and marked nephritis with œdema after the fever has subsided has been recorded. Relapses are rare.

The pathology of mumps is not well understood. Laveran and Catrin found in the blood, and in the affected parotids and testicles a micrococcus—usually in pairs—which could be cultivated at 35° C., but inoculations in animals were negative. The analogy which epidemic parotitis presents to the parotitis which occurs as a complication of intraabdominal suppuration, has led to the view that the inflammation arises in the ducts, but such anatomical evidence as exists points to the interstitial tissue as the part affected. When the testicle atrophies after orchitis the whole organ becomes soft, the seminiferous tubules lose their epithelium, and there is an overgrowth of connective tissue.

The diagnosis is usually easy, as parotitis from other causes is very rare in childhood. Enlargement of the lymphatic glands behind the angle of the jaw (secondary usually to tonsillitis or prolonged stomatitis) is often spoken of by parents as mumps, but observation of the situation of the swelling behind and in front of the ear, which is displaced outwards so that the lower part of the auricle stands out, ought to prevent the possibility of a mistake. The chief risk of error is that the disease may be overlooked if the parotid swelling is slight, and an epidemic thus permitted to start in a school. Prophylaxis is rendered difficult by the early commencement of infection. If the patient be isolated in a room apart from other children,

the infection will not as a rule spread beyond those already infected. Infection may have taken place in any children who have been in contact with the patient during the four days previous to the parotitis. A child who has been exposed to infection should not be allowed to mix with other susceptible children, as, for instance, in a school, until twenty-five days after its exposure ceased.

Treatment cannot arrest the course of the disease. While the temperature is raised the patient should be kept in bed, and at the onset he should take a purge. Hot applications to the swollen part are grateful to the patient at first, and later a cotton-wool pad should be applied. The mouth and pharynx should be kept clean by the use of gargles, lotions, and sprays. Very acute swelling and pain should be treated by belladonna fomentations or belladonna and glycerine smeared on the part and covered with cotton-wool. High temperature and delirium call for sponging with cool water and the ice cap, while quinine and antipyrine may be given together or separately. The food should be light, and at first fluid only. The tendency to constipation is often troublesome, and should be met by the exhibition every morning or every other morning of a simple laxative, such as liquorice powder.

Glandular fever was described by Pfeiffer in 1889[1] as a fever attended by adenitis and due to specific infection.

The disease has been seen in infants as young as seven months, and in children of thirteen years, but the majority of the cases occur between the ages of four and seven years.

The infection, of which the agent has not been isolated, is little diffusible, but most of the children in a family suffer. J. P. West has described recently[2] an epidemic which spread very slowly through a thinly inhabited district of Ohio. The period of incubation is probably about seven days.

The child is taken ill suddenly with headache, pain and stiffness in the neck, some pain on swallowing, and often general pains in the back and limbs, which may suggest the onset of rheumatism. At the same time the temperature rises to about 102° F., the pulse becomes rapid, and respiration is quickened. The face is flushed, but there is no rash. On the second or third day a swelling is noticed behind the angle of the jaw, and extending along and beneath the sternomastoid muscle. On palpation it is found to consist of three or four glands, which are enlarged, firm and tender. In about two days this adenitis, which begins usually on the left side, reaches its height, and the corresponding glands on the other side then begin to enlarge. Other cervical and the axillary and inguinal glands may be affected. Pain in the abdomen is often present, and

[1] *Jahrb. f. Kinderhlde.*, Band xxix., s. 259.
[2] *Arch. of Pediatrics*, Dec., 1896.

in a considerable proportion of cases the mesenteric glands are enlarged. The child is thirsty, but has no appetite; the tongue has a white coating, and there is constipation in all but the mildest cases. The spleen and liver are usually enlarged. There may be a little redness of the pharynx and tonsils, but the latter are not enlarged as a rule. In a small number of cases there is severe pharyngitis. The temperature reaches its highest point (104° F., or higher) at the time the swelling on the side first affected is at its height. It may then fall considerably, to rise again as other groups of glands are involved. The final defervescence occurs from a week to a fortnight after the onset; it may be rapid and accompanied by the passage of green mucoid stools. The glands suppurate very rarely, if ever, but remain enlarged for some days or weeks after defervescence. Convalescence is often slow, owing to anæmia and general depression. Complications are rare, but acute nephritis may occur.

The prognosis is good, and very few deaths have been recorded.

The diagnosis must usually be difficult, as doubts as to the specificity of the adenitis may well be entertained. A similar train of symptoms may attend adenitis secondary to obvious lesions of the mucous membranes or skin. Neumann believes that the active agents are streptococci or staphylococci which have passed through the tonsils and pharyngeal mucous membrane without producing local lesions; but against this view is the rarity of suppuration. It has been suggested that the infection finds entrance through the gastro-intestinal mucous membrane, and that the glands on the left side of the neck are affected first, owing to their contiguity to the thoracic duct. In two epidemics of adenitis of the subauricular and submaxillary lymphatic glands the disease was shown to be, in reality, mumps, by the occurrence of parotid swelling in some cases; and this possibility should be borne in mind. It is said also that rubella may occur in epidemics, in which there is no rash, though the glandular swelling is well marked.

The treatment should consist in keeping the child in bed on a fluid diet. The pain and stiffness in the neck may be relieved by belladonna liniment, belladonna and glycerine, or by cold compresses. Constipation should be treated by mild laxatives or by enemata, and afterwards salol, naphthalin, or small doses of calomel should be given. Purgation does not cut short the attack, and tends to increase the subsequent depression.

Cerebro-spinal meningitis is an acute infective disease which occurs sporadically and in epidemics.

Numerous epidemics have occurred in Germany and in North America, very few in Great Britain, where, however, sporadic cases are not uncommon. In some epidemics children have been attacked in much greater numbers than adults; in others the reverse has been

the case; while in others, again, all ages have suffered to about an equal extent. Epidemics appear to depend upon local conditions, the nature of which has not been ascertained. Direct infection has not been proved to occur.

The pathology of the disease is obscure, and the infective agent has not been identified. [The *diplococcus intracellularis meningitidis* described by Weichselbaum,[1] Jager,[2] Councilman,[3] Mallory,[4] Wentworth,[5] and others, is at the present writing universally conceded to be the cause of epidemic cerebro-spinal meningitis.

It is a diplococcus found inside the pus-cells in the meningeal exudate, in the spinal fluid obtained by lumbar puncture, in the nasal secretions and in the purulent discharge of an otitis occurring as a complication.] There is a general meningitis affecting the membranes both of the cord and of the brain, with extreme congestion of the brain and cord, accompanied sometimes by actual hæmorrhage or disseminated areas of encephalitis. On the surface of the cerebral and spinal membranes purulent exudations may form, and there is some effusion into the ventricles. In more chronic cases the meningitis is plastic, characterized by adhesion and thickening, and the effusion into the cerebral ventricles may be considerable. Pneumonia, pleurisy, endocarditis, and nephritis may occur as complications. In severe cases extensive hæmorrhages into the skin and serous membranes may occur early, and the patient may die before the meningeal lesions have become well marked.

The symptoms usually come on suddenly, or there may be for some days headache, backache, and malaise. The earliest symptoms are headache, shivering, rigor or convulsions, and rise of temperature to 101° or 102° F. The headache increases, the neck becomes stiff and painful, there is photophobia and dread of noise, and great restlessness and irritability. The stiffness of the muscles of the neck passes on into extreme rigidity, so that the body is stiff like a statue, or there may be extreme retraction of the head. Pain in the back and limbs is present, and may be very severe, and there may be spasm, clonic or tonic, of the limbs and of the face. Strabismus is a frequent symptom. In addition to the pain in the back and occipital region, there may be hyperæsthesia along the spine. At the onset there may be convulsions or furious delirium, which, as the effusion increases, gives place to somnolence, and finally to coma. The pulse is usually very rapid, but the respirations are not much hurried, and may be slow or present the Cheyne-Stokes character. The temperature may not rise much after the first elevation at the

[1] Weichselbaum: *Fortschritte der Medicine*, Bd. 5, 1887.
[2] Jaeger: *Zeitschrift f. Hygiene u. Infectionskrank.*, Bd. 19, 1895.
[3] Councilman: *Trans. Assoc. Amer. Physic.*, 1897.
[4] Mallory: Pathological Technique.
[5] Wentworth: *Boston Med. and Surg. Journal*, Vol. CXXXVIII., No. 11.

onset, or it may fluctuate very much, or it may show a steady rise, reaching 106° to 108° F. before death. Herpes is extremely frequent, and rose-colored spots like those of typhoid, urticaria, erythema nodosum, and ecthymatous and pemphigoid eruptions are among the various rashes which have been observed; but the most common skin lesion is hæmorrhage into the skin. Sometimes petechiæ and purple spots are very numerous and cover almost all parts of the skin. The bowels are usually constipated, but there may be diarrhœa. Vomiting, which usually occurs at the onset, is not a prominent symptom subsequently. The urine may contain albumen, and in the most acute cases blood. Death may occur in so short a time as twenty hours, before the development of characteristic symptoms, or the case may run a subacute course lasting many weeks, or even months, and eventually end in recovery. Usually, however, if recovery is to take place, improvement begins between the fourth and sixth day. Pneumonia is the most important complication, and blindness from optic nerve atrophy, and deafness from labyrinthine disease the most serious sequelæ.

The prognosis is uncertain. In severe cases, with petechiæ and extensive rigidity, it is bad. Herpes is also an unfavorable sign. The death-rate varies very much in different epidemics. It may be as low as 2 or 3 per cent., or as high as 75 per cent. The disease is generally more severe in children than in adults. The diagnosis may be impossible in sporadic cases, since the symptoms closely resemble those of tuberculous meningitis. Well-marked rigidity, the occurrence of herpes, a regular pulse, and the absence of the peculiar soft feeling of the abdomen usual in tuberculous meningitis, may point to the true diagnosis. When both pneumonia and cerebro-spinal meningitis are present, it may be impossible in sporadic cases to determine which is the primary disease. Cerebro-spinal meningitis presents often a great resemblance to typhoid fever with pronounced cerebral symptoms; and if the symptoms of the former are not well marked, diagnosis may be quite impossible, since enlargement of the spleen may or may not be present in both.

[Most authorities[1] concede that a positive diagnosis can be made by means of lumbar puncture. It consists in tapping the spinal canal in the lumbar region and examining the fluid macroscopically, microscopically, and bacteriologically. Thus we can always detect the presence of a meningitis by a greater or less degree of turbidity of the fluid, normal spinal fluid being clear. Also we can often by further examination of the fluid and its sediment, by cultures and inoculation experiments, determine the kind of meningitis present, cerebro-spinal, tubercular, etc. In the cerebro-spinal variety, the

[1] For technique, see Wentworth, *Trans. Amer. Pediatric Soc.*, Vol. VIII., 1896, and Wentworth, *loc. cit.;* also page 534, below.

fluid obtained is more or less turbid, and contains some sediment, cover-glass preparations of which show "numerous polymorphonuclear leucocytes," "pus-corpuscles," occasional smaller mononuclear lymphoid cells and fibrin. Groups of the diplococcus intracellularis are found in varying numbers in the protoplasm of some of the leucocytes.]

The treatment must be symptomatic. The severe headache and stiffness in the neck may be relieved by dry cupping, and by the application of ice-bags to the head. Of internal remedies for the spasm, morphia, either by the mouth or hypodermically, is the most efficacious, but bromides are also useful. Potassium iodide has been thought to exercise a beneficial effect on the meningitis. The patient should be carefully fed, if necessary by the stomach tube.

Typhoid fever (*enteric fever*) is an acute specific disease due to infection by the bacillus typhosus, an organism which resembles closely the b. coli communis.

The bacillus is localized mainly in the lymphoid tissue of the small intestine, especially in Peyer's patches, where it produces a specific inflammation; but it may become established secondarily in other organs. The intestinal inflammation may terminate in sloughing and ulceration, or in resolution, which occurs more often in children than in adults. The infection is disseminated usually by water, sometimes by milk or cream which has become contaminated by water specifically polluted, more rarely by contaminated utensils or uncooked vegetables.

Typhoid fever is a milder disease in children than in adults, its course shorter, its symptoms less severe, its mortality lower. The severity increases directly with age,[1] and is greater at ages over than under ten years. It is as common between five and ten as between ten and fifteen. It is probably very rare in infants, and is seldom recognized under two years of age, a period of life when the symptoms are extremely mild. The proportion of children infected during an epidemic varies, but is often high when the infection has been distributed by milk.

The *incubation period* varies within rather wide limits. It is most often twelve to fourteen days, not infrequently nine or ten, occasionally less. It probably never much exceeds three weeks. Infection may be derived from a patient during the whole course of the fever, and for the first fortnight of convalescence. It may be retained by fomites for two months at least.

The *pyrexia* of typhoid fever may be divided into two periods; (1) The period of primary or specific fever, corresponding to the invasion and establishment of the disease, during which the specific inflammation of the lymphoid structures of the intestines takes place;

[1] J. L. Morse, *Bost. Med. and Surg. Journ.*, Feb. 27, 1896.

and (2) the period of secondary or suppurative fever attending the formation and separation of the intestinal sloughs and the consequent ulceration.

The *onset* is more often acute in children than in adults, and in children under ten than in those over; yet in from half to two-thirds of the cases in children the onset is insidious. The earliest symptoms may be shivering or a rigor, more often vomiting. The temperature rises at night and falls in the morning, the morning fall being less and the evening rise greater for five or six days, until the maximum is reached. The temperature continues to show fairly regular oscillations, morning fall and evening rise, for about a week. With the development of the secondary period the oscillations gradually grow wider, the remissions being more marked and the evening maxima somewhat less high; the range of the diurnal oscillation becomes gradually less, until finally, after a variable period, three to five weeks after the onset, the normal is reached. When the onset is sudden, the maximum may be reached within the first two or three days. During the period of primary fever the oscillations are often much greater than in adults. Owing to the frequency with which resolution of the intestinal inflammation occurs without suppuration, secondary fever is in children absent in many, probably about half, the cases. The average duration of the fever is less than three weeks, and in children under ten is often much shorter—less than two weeks. The pulse is soft and increased in rapidity, but not in proportion to the height of the temperature. A systolic apex murmur is heard in many cases, but disappears during convalescence, and marked cardiac weakness may occur during the fever or during convalescence.

The *symptoms* are commonly not well marked in children, and the younger the child the less characteristic are they. Diarrhœa is absent in a large number, probably the majority, of cases, and constipation may be a troublesome symptom. Morse records diarrhœa in 32.5 per cent. from five to ten years, and in 42 per cent. from ten to fifteen years; but it was severe in only 2.6 per cent. at the earlier, and 2 per cent. at the later age. Tympanitic distension of the abdomen is common, but tenderness is often very little marked, especially in children under ten. Enlargement of the spleen is the rule; it is often slight, but in young children may be very considerable, especially in the early stage. The tongue is tremulous; it may be clean, but it is usually thickly coated with a cream-colored fur. Dryness of the mouth and tongue, and cracked lips, are far less common than in adults. Hæmorrhage from the intestines is comparatively infrequent and perforation extremely rare.

A roseolous *eruption* occurs as in adults in most cases; but it is usually scanty, and often disappears rapidly. Other eruptions are

rare, but sudamina, maculæ, petechiæ and ecchymoses, urticaria, and labial herpes may occur. Boils, sometimes in large numbers, may cause much discomfort during the later stage of the attack and in convalescence. Bronchitis is a less frequent, but, when present, a more prominent symptom than in adults. Some bronchial catarrh occurs in from a third to half the cases, but severe bronchitis is most common in young children. Broncho-pneumonia and pleuro-pneumonia are not uncommon. Acute pharyngitis may produce marked symptoms at the onset, and laryngitis occurs in a considerable proportion of cases in some epidemics. The face is dusky, and wears an expression of depression and lassitude. The patient lies on his back in bed, and appears to wish only to be left alone. The severity of the nervous symptoms varies greatly; not infrequently they are very slight, though headache, not usually severe, is present in the majority of cases. It disappears with the onset of delirium, which is usually mild and wandering, but sometimes noisy at night. In a small number of cases—the proportion being larger under ten years—there are marked nervous symptoms, suggesting meningitis—retraction of the head, opisthotonos, pain and tenderness in the neck, photophobia, inequality of the pupils. Suppurative otitis media is a not infrequent complication, and may be attended by meningeal symptoms, pain causing fits of screaming, delirium, and high temperature. Epistaxis is common, but is seldom severe. Albuminuria is frequent, but true nephritis is said to be rare. [A changed mental condition sometimes occurs during convalescence. Thus some children show a marked irritability or moodiness, others again are extremely forgetful, have less power of concentration and are easily fatigued by any mental exertion. Such children should be carefully watched as to their school-hygiene and no excess in studies allowed.]

The diagnosis may be very difficult, owing to the absence of characteristic symptoms. If in the early stage bronchitis or pneumonia be present, all the symptoms are very apt to be attributed to these complications. Even after death, bacteriological examination alone may suffice to determine whether the swelling of Peyer's patches and the mesenteric glands is specific. At a later stage the continuous fever, coated tongue, dusky face, and abdominal tenderness may enable a diagnosis to be made even in the absence of characteristic diarrhœa. When nervous symptoms are prominent, the resemblance to tuberculous meningitis may be close, and acute general tuberculosis may be mistaken for typhoid fever (see "Tuberculosis," p. 168). Such cases have been mistaken also for epidemic cerebro-spinal meningitis, the error being discovered only *post-mortem*. In malarial regions the autumnal type may present a striking similarity in its early days to typhoid fever, and the diagnosis may be possible only

by the discovery of the malarial parasite in the blood (Osler). In future the serum method of diagnosis will probably be of great use in those cases in which it is available.

[Recent research has conclusively demonstrated the Widal serum test to be of practical value in many cases. The reaction when found is proof positive of the presence of typhoid: a negative result is of little diagnostic significance. The reaction is found as early as the seventh day, disappearing a varying length of time after the cessation of the disease.]

The prognosis, as already indicated, is more favorable in children than in adults. Extreme tympanites, especially if accompanied by vomiting, is a bad omen, as is also the early onset of nervous symptoms or great depression. Bleeding from the bowel, if small in quantity, is not necessarily serious, but if repeated frequently, or very copious, indicates serious ulceration and imperfect repair.

In treatment the main indication is rest in bed. Good nursing is essential, and special care should be taken to keep the patient clean and free from bed-sores. Milk should form the main part of the food, but it should be given diluted, and the effect on the comfort of the patient and the condition of the stools watched, since the thirst from which the patient suffers may easily induce him to take more milk than can be digested. A mineral water containing a low proportion of carbonic acid is a good beverage, or water acidulated with hydrochloric acid, citric acid, or lemon juice. When constipation exists vegetable soups may be tried, or a small dose of castor oil may be given.

The cold bath treatment has not yielded satisfactory results as a routine measure, and the use of warm baths cooled down by the addition of ice or cold water is only called for in cases in which the temperature remains elevated for an unusual time. On the whole, the best results have been obtained by the most simple means. When diarrhœa is severe, which is not often the case, it may usually be checked by an enema of starch and opium. As a rule, it will be found to be due to the presence of curds or other irritating remnants of food, and to moderate as soon as the diet is regulated. Hæmorrhage from the bowel will be treated on the same principles by diminishing the amount of food, by allowing the patient to suck ice, and only in severe cases by the administration of acetate of lead and opium. Tympanites may be relieved by the application of turpentine stupes. In the management of convalescence the safest rule is to permit no solid food until ten days after the temperature has become normal, and to keep the patient in bed for this period.

[Great care should be taken in the disinfection of all excreta. If the discharges be in a vessel, they should be covered with a solution of carbolic acid 1 : 20 for a period of six or eight hours before being

thrown away. All napkins should be similarly treated and then boiled, or if possible burned. The bed linen should be boiled separately from the family wash.

The investigations of Richardson (Boston) in the urine of typhoid adults are of great practical importance. He has demonstrated the presence of typhoid bacilli in the urine of convalescents for weeks and even months after the acute stage, and states that their elimination is hastened by the administration of urotropin. He advises giving this drug, 30 grains daily, for 10 days, beginning with the third or fourth week of the disease.]

CHAPTER VIII.

ACUTE SPECIFIC INFECTIOUS DISEASES (*concluded*).

Diphtheria—Incubation Period—Pathology—Symptoms—Diphtherial Palsy—Diagnosis—Prognosis—Antitoxin Treatment—General and Local Treatment—[Immunization].

Diphtheria is a specific inflammation affecting the mucous and cutaneous surfaces, produced by a specific bacillus, and characterized by the formation of membranes.

The interval between exposure to infection and the development of characteristic symptoms is variable; it is most often two days, and does not as a rule exceed four days. *Infection* may be derived from a patient suffering from diphtheria in the incubative stage, during the attack, for a period of long and probably varying duration after apparent recovery, from fomites, or from contaminated milk. It may be derived from mild or anomalous unrecognized cases.

The *diphtheria bacillus*, called after its discoverers the Klebs-Löffler bacillus, grows readily on blood-serum containing glucose and bouillon, but also on other culture media. It flourishes best at 98° to 101° F., forming elevated grayish-white colonies with opaque centres, which first become perceptible about fifteen hours after inoculation of the tube. The bacillus itself, which is not motile, and is not known to form spores, is 2.5 to 3 μ long, and about one-fifth of this in breadth; it is slightly thickened at each end and curved. It varies very much in virulence, some specimens being harmless. The less virulent bacilli (pseudo-diphtheria bacilli) are usually shorter and straighter, and grow more freely at low temperatures (68° F. or less). The bacillus is very resistant to drying, and its virulence when attenuated, but not suppressed, may become restored. In a state of little or even of considerable virulence the bacillus may be present in the throat or nose without producing any lesion, and it is probable that it can only attack the epithelium when this has been damaged. The presence of the streptococcus pyogenes in association with the diphtheria bacillus appears to exalt the virulence of the latter. The streptococcus also by attacking the epithelium may produce a lesion which will enable the diphtheria bacillus to establish itself. This

may account for some of those cases of, often, very virulent diphtheria which arise without any discoverable source of infection, after exposure to cold or as a complication of scarlet fever, measles, typhoid fever, and other acute diseases.

The bacilli, once enabled to attack the mucous membrane or skin, kill the epithelial cells, and excite inflammation with effusion of fibrin and migration of leucocytes, which are likewise killed in greater or less number; after a time a patch of false membrane is thus produced. At the periphery of the membrane the epithelium is proliferating, and infiltrated with white and red corpuscles and fibrin. At the focus the epithelium is replaced by false membrane, which consists of a fibrinous exudation, in the meshes of which are contained, at the surface, great numbers of micro-organisms, usually, in addition to the diphtheria bacillus, streptococci and staphylococci, beneath this, fibrin and degenerating cells with a few microbes, and, deeper, epithelial cells, and many leucocytes enclosed in irregular meshes of fibrin. The bacillus may be conveyed from the original point of infection to other parts (1) by contact, as when one tonsil becomes infected from a false membrane on the other; it may spread (2) along passages—the bronchi, Eustachian canal, the œsophagus, or into the nose—with or without the formation of visible false membrane; (3) along the lymphatics to the glands; and (4) at the approach of death, and possibly under other conditions it may be found in the spleen, liver, and kidneys, to which it must have been carried by the blood. Diphtheria, especially when it involves the larynx, is very frequently complicated by broncho-pneumonia (or bronchitis with collapse). In such cases, the bacillus diphtheriæ is present in the lungs; it is associated with other microbes, but it is probable that it can itself produce broncho-pneumonia. The general symptoms of diphtheria are due to the absorption of soluble bodies,[1] which have a poisonous action on the leucocytes, and on the cellular elements of the organs (*e. g.*, the kidneys, producing glomerulo-nephritis), but has apparently a selective action on the nervous system. At the same time it causes a fall of blood-pressure and dilatation of the vessels, especially those of the lungs, liver, and kidneys, and diminishes the force of the heart. The blood contains an excess of leucocytes, is altered in color, and does not coagulate firmly. The severity of the toxic symptoms is not in direct proportion to the extent of the false membrane, but is dependent in part on the idiosyncrasy of the individual, in part on the nature of the bacillus, some types apparently producing more toxin than others—and in part on the extent to which the lungs are involved. The fact that in severe cases the lungs so often contain the bacillus diphtheriæ is, it will be

[1] Roux and Yersin, *Annales de l' Institut Pasteur;* Sidney Martin, *Brit. Med. Journ.*, 1892, vol. i., p. 641 *et seq.*

seen, of importance in this connection, since it would seem that the quantity of toxin produced may thus be very greatly increased.[1]

True diphtherial inflammation may be complicated by the presence of various pyococcal organisms. Inflammation of the fauces, due to the streptococcus pyogenes, may become infected by diphtheria—an event of not infrequent occurrence in scarlet fever—or a true diphtherial inflammation may be complicated from the first or at a later stage, by the streptococcus, more rarely by the staphylococcus.[2] The combination of the streptococcus with the diphtheria bacillus produces, as a rule, an affection severe both in its local and general manifestations; but this is not always the case, and in some instances of mixed infection the course of the disease does not differ from that of uncomplicated diphtheria.

[The cardiac weakness so often seen in diphtheria would seem to be due to degenerative changes both in the myocardium and in the nerve fibres of the vagus. Thomas and Hibbard[3] have studied these changes exhaustively and attribute sudden death to the effect of the diphtheria toxin on the nerve structures of the heart. They also call attention to the opinion that the innervation of the levator palatæ and azygos uvula is now thought to be by the vagus and that hence all patients with palatal paralysis should be kept in bed, whether or not there be any evident change in the pulse or heart action, on account of the possible danger of sudden cardiac failure.]

Symptoms.—The onset of diphtheria may be acute or insidious. In the former case the child becomes suddenly ill, complains of cold, shivers, perhaps vomits, or has a convulsion. It is then found that the temperature is raised to 102° or 103° F., and that the child is drowsy, and has headache and pains in the limbs. At this time no false membrane may be discoverable; but if the *pharynx* is to be its seat, there will be some redness and tumefaction of the mucous membrane, swelling of the tonsils, and tenderness over the glands behind the jaw; or if the larynx is the primary seat of the infection, there will probably be some slight hoarseness, which, in association with the general depression, will excite a suspicion of diphtheria. In the insidious cases advice is usually not sought until the child has suffered for some days from lassitude, depression, and loss of appetite, although there may already be extensive membranous inflammation in the throat. Not infrequently the first case in a family has this insidious onset, and its existence is only discovered when

[1] A paper by Kanthack and Stephens should be read in this connection; *Journ. Path. and Bact.*, vol. iv., p. 45.

[2] Hewlett and Nolan (*Brit. Med. Journ.*, vol. i., 1896, p. 266) found, out of a total of 353 cases, the diphtheria bacillus pure in 216, associated with the streptococcus alone in six only, and with the streptococcus and other microbes in thirteen others.

[3] Med. and Surg. report of Boston City Hospital, 9th series, 1898.

medical advice is sought for another child, in whom the attack, contracted from the first, has begun suddenly. In some cases, with insidious onset, the first symptom to attract attention is swelling of the neck, due mainly to adenitis. Pain in the throat may not be an early, nor at any time a prominent, symptom. In other cases dysphagia is the earliest symptom—the tonsils are enlarged and the fauces red and swollen, or œdematous. This condition of apparently simple inflammation may persist for several days before membrane forms. In some few cases of pharyngeal diphtheria no distinct membrane is seen at any stage of the case, either because it is not formed, or because it occupies a site not open to inspection. In other cases the appearances very closely resemble those of follicular tonsillitis. The *false membrane*, which usually appears first on the uvula, the edge of the soft palate, or the tonsils, is at first thin and semitransparent or opalescent. Later it becomes thick, and of an opaque white or faintly yellow color. In consistency it varies, being sometimes tough, at others friable, but tending to be tough at first and friable as recovery begins.

The *extent* of surface covered by the membrane, and the rapidity with which it spreads, varies in different cases. A small patch on the tonsil or soft palate may have spread on the second day to the whole of the soft palate, tonsils, and pharynx, and extension may also have taken place into the nose and larynx. If removed mechanically, it is quickly re-formed. The swelling of the surrounding mucous membrane is usually in proportion to the acuteness of the local process. After forty-eight to seventy-two hours the membrane usually becomes detached, sometimes in flakes. In other cases it undergoes rapid decomposition, giving rise to a fœtid odor and a brownish, sometimes blood-stained, secretion. In such cases deep ulceration may follow the detachment of the membrane. As a rule, when detached spontaneously, it is not reproduced, and, in mild cases, the mucous membrane quickly returns to its normal color, while the swelling disappears more gradually. Some adenitis at the angle of the jaw is the rule in even mild cases of diphtheria. Its extent is proportionate to the extent of surface involved by the membrane, and is only so far proportionate to the severity of the attack. In the most severe cases, with early toxæmia, there may be little adenitis. The membrane seldom affects the cheeks. The tongue is often thickly furred, though not the seat of membrane. The temperature usually falls soon after the onset, and, during a moderately severe attack, may not again rise above 101° F. In the most severe cases, in which toxæmic symptoms are prominent, the temperature may be subnormal. The pulse is accelerated in proportion to the temperature, but in toxæmia it becomes small, weak, and irregular.

Diphtheria may cause death, or extreme risk to life, in several

ways. Of these the most frequent are: (1) The obstruction to respiration produced mechanically by laryngeal diphtheria; (2) broncho-pneumonia and bronchitis, with collapse; (3) diphtherial toxæmia, or the combination of this with septic toxæmia; (4) paralysis (*a*) of heart and respiration, which may occur early in convalescence or before it is established, or (*b*) general paralysis, involving eventually the respiratory or cardiac systems.

The *larynx* is affected in about one-sixth of all recognized cases of diphtheria,[1] and the mortality is high (over 50 per cent.). The younger the child, the greater the danger to life. The affection of the larynx may be primary; more often it is secondary to pharyngeal diphtheria. In considering the symptoms of laryngeal diphtheria, it is useful to bear in mind the classification of Barthez, although it is not possible in all cases to mark the several stages. In the *initial* stage the voice is hoarse, as is also the cough, which comes on often in paroxysms, ending in the expulsion of a little mucus. The inspiratory murmur over the chest is harsh, but there is no obvious laryngeal obstruction. The *spasmodic* stage ensues after an interval of varying, but usually short, duration. Respiration becomes slightly embarrassed; inspiration is prolonged, and often accompanied by slight stridor and by recession in the suprasternal notch and at the epigastrium. The face is pale, a little dusky, the eyes prominent and glassy. The child is very restless and peevish. Presently it has an attack of suffocative dyspnœa on waking from sleep, or after coughing, crying, or swallowing. The attack ends, perhaps, in a severe coughing fit, followed by the expulsion of glairy mucus or a fragment of membrane. In favorable cases there may be only one or two such attacks, but in severe cases the intervals between succeeding attacks become shorter, until finally a condition of permanent dyspnœa is established. In this—the stage of *mechanical obstruction*—inspiration is noisy, prolonged, and attended by extreme recession of the epigastrium, the lower part of the sternum, and the attached ribs; expiration is short; and the pause after expiration is absent. The pulse is weak; during inspiration it becomes more rapid, and almost, or quite, imperceptible at the wrist (*pulsus paradoxus*). The child is less restless—the face more pale or dusky; the lips purple; the eyes prominent, fixed and glassy. There is, in fact, a condition of partial asphyxia, by which eventually consciousness is dulled, and the child dies asphyxiated. Laryngeal diphtheria may be complicated by membrane in the trachea and bronchi; more rarely these parts are infected before the larynx. The diagnosis is difficult, as the symptoms suggest the onset of broncho-pneumonia; respira-

[1] In the Metropolitan Asylums Board's hospital in 1894–5 there were 6,571 cases of diphtheria. Of this number 1,009 suffered from laryngeal diphtheria, of whom 519 died.

tion is hurried; recession is not marked, but the face is pale or cyanosed. In some cases casts of the trachea and bronchi have been coughed up.

Broncho-pneumonia may complicate pharyngeal diphtheria, but is far more common as a complication of laryngeal diphtheria. It comes on either early, during the first two or three days, or a few days after tracheotomy. It is a secondary affection, due, probably, in the majority of cases, to infection by the streptococcus, but, as has been observed above, the diphtheria bacillus may be present also. Bronchitis is seldom in itself an important complication of diphtheria, but it favors collapse of the lung in children with soft chest walls, which is not only dangerous in itself, but favors the onset of pneumonia. The occurrence of broncho-pneumonia causes a rise of temperature, accompanied by marked increase in the respiration rate.

When *toxæmia* is the cause of death, it is produced either by the severity of the diphtherial infection or by its association with some other infective agent, usually the streptococcus. In the former case, toxic symptoms may exist almost from the onset of the disease, or come on at the commencement of convalescence. There is a rise of temperature, and the child sinks into a condition of great depression. The face is pale or leaden, the lips cyanosed, the eyes are sunken, and there is complete loss of appetite; yet there is no dyspnœa. When the toxæmia is due to an association of the streptococcus with the diphtheria bacillus, septic or malignant diphtheria, the general symptoms are usually, from the first, of marked adynamic type. In its most acute form this is an exceedingly fatal disorder. There is much tumefaction of the pharynx; the false membranes are voluminous, soft, and bleed easily; the cervical glands are involved early, and swell to a great size. The nasal mucous membrane is usually infected, and the foul and infective secretion from the nostrils leads to excoriation and secondary infection of the upper lip and other parts of the face. The prostration is extreme, and the patient, in most cases, succumbs in two or three days. Cases of less severe type also occur, which run a less rapid course, in which the general prostration is less severe, and in which, consequently, there is more hope that treatment, if applied early, may avert a fatal termination.

Cardiac failure is one of the most common and distressing causes of death during the early stage (fourth to tenth day) of cases in which the larynx is spared. At this stage it is due probably to degenerative changes produced in the muscular substance of the heart by the toxins. The symptoms are not very well marked; there is pallor, debility, or prostration, which increases gradually; the pulse is small, soft, irregular; the cardiac impulse is feeble, the first sound soft, toneless, the second often reduplicated. Slight exertion produces dyspnœa, and the patient may die suddenly in attempting

to get out of bed, or even in the act of sitting up. A very similar train of symptoms may be observed at a later stage, but sometimes associated with sudden attacks of dyspnœa, and attended by vomiting. In such cases it is probable that there is a neuritis of the vagus. Not very infrequently sudden death occurs early in the convalescent stage after some trifling exertion, and it is apparently due to this cause. More often, however, cardiac failure occurs later in patients who have already suffered from more or less widespread paralysis or ataxy.

Reference has already been made to the infection of the nose in cases of toxæmic diphtheria, but the *nasal* passages may be the seat of uncomplicated diphtheria. In such cases, which are rare, the membrane, present on one or both sides, is usually thick, the discharge from the nostrils is serous or muco-purulent but scanty, and the general symptoms are not severe. Usually such cases run a mild course, but occasionally the larynx becomes infected.

Diphtheria of the *conjunctiva* may be primary, or secondary to nasal diphtheria. In cases of moderate severity there are the customary symptoms of severe conjunctivitis, but the palpebral conjunctiva is found to be covered by a thin false membrane. In milder cases no false membrane is produced and the diagnosis must rest on the probability of infection, or on the results of bacteriological examination. The most severe form, which occurs as a complication of toxic nasal diphtheria, is very grave in its local results; extensive interstitial inflammation produces a kind of solid œdema of the eyelids, and ulceration of the cornea ensues, with the result that even if perforation be escaped, corneal opacities and adhesion of the conjunctiva still remain.

Diphtheria of the mucous membrane of the *mouth* is rare. The *vulva* is occasionally infected, usually as a complication of measles or scarlet fever; the false membrane forms, as a rule, on the inner aspect of the labia majora, and the anus may be affected secondarily. Very rarely is the prepuce or glans in boys the seat of diphtheria.

Primary diphtheria of the *skin* is a rare accident, but it is not uncommon to see excoriations or ulcerations about the nose or mouth infected secondarily in toxic (septic) cases.

Many of the *complications* of diphtheria have already been mentioned incidentally.

Diphtherial palsy occurs in about one-fifth of the cases which survive the acute attack. It varies much in extent, so that two forms are usually distinguished—local and general. Paralysis begins usually in the second or third week, and, therefore, after the false membrane has cleared away and convalescence appears to have commenced. It may commence, however, during the course of the attack. When it appears early it begins almost invariably in the

soft palate, is often limited to it, and would seem to be a local process. Even in such cases, however, the knee-jerks usually disappear, but they may be absent also during convalescence in cases in which no palsy is observed at any time. Paralysis of the soft palate is indicated by immobility, or diminished mobility of the soft palate; by the "nasal tone" of the voice; and by the return of fluids through the nose when an attempt is made to swallow. In other cases the palsy extends to the pharynx, and there is added to the other symptoms a difficulty in swallowing, and a risk of the entrance of particles of food into the larynx producing suffocative attacks, and possibly pneumonia. Apart from an accident of this nature, however, the prognosis in these limited cases is good, and wide extension of the paralytic symptoms is rare. The entrance of food into the air passages is greatly favored by palsy of the upper constrictors of the larynx, which, however, occurs less frequently than paresis or paralysis of the glottis closers. This defect causes the voice to be whispering, and deprives the cough of its explosive character, thus rendering it ineffective and hindering the expulsion of mucus. Weakness of the muscles of the mouth is sometimes associated with the palatal palsy, rendering sucking and even mastication difficult, but definite facial paralysis of one side may also occur.

Ophthalmoplegia, externa or interna, is not uncommon. It is usually an early, and may be the only, symptom of diphtherial palsy, though it is sometimes followed by palatal paralysis. Ophthalmoplegia interna may affect the ciliary muscle (cycloplegia). This causes in an emmetropic eye indistinctness of near vision, in the myopic eye very little disturbance of vision; but in the hypermetropic eye, in which the focus of the lens system lies behind the retina so that some contraction of the ciliary muscle is needed, even for parallel rays (*i. e.*, for distant vision), the failure of the ciliary muscle may practically destroy useful vision. In accommodation for near objects, convergence and contraction of the pupil are associated movements. In diphtherial palsy there may be loss of myosis on convergence, or of convergence and myosis. More rarely the pupil fails to react to light. Ophthalmoplegia externa, which is rarer than ophthalmoplegia interna, is in many cases associated with evidence of involvement of the cardio-respiratory centres. The occurrence of strabismus therefore adds to the gravity of the prognosis of diphtherial palsy far more than the onset of internal ophthalmoplegia. Degenerative changes, and capillary hæmorrhages into the pons, and the gray matter of the fourth ventricle have been recognized in cases examined after death.

Those forms of diphtherial palsy to which the term *generalized* is applied do not differ essentially from the more limited forms, except in the rapidity with which many parts are invaded; the muscles of

the head and neck, or of the lower extremities or the cardio-respiratory system are, in various cases, the parts earliest or most seriously involved. In the second or third week the child begins to have some difficulty in swallowing, and to speak with a nasal tone. Soon its face assumes an expression of listlessness, owing to weakness of the facial muscles, and the head falls forward owing to failure of the posterior cervical muscles. In other cases, the first thing noticed is that there is weakness of the lower extremities, and the child soon becomes unable to walk or to stand without support. This inability is due to paresis, but in many cases is aggravated by ataxy. In other cases, again, *ataxy* is the first symptom, or it becomes associated at an early date with the palsy of cervical muscles. *Cardio-respiratory* paralysis may develop independently or in association with cervical palsy. Complaint is made of abdominal pain, which is followed by vomiting; there is slight dyspnœa, and the pulse is slow. Gradually the respiration becomes more rapid, irregular, or sighing; the pulse also rapid; the face pale and anxious. A fatal attack of dyspnœa—spontaneous, or determined by some slight exertion, or by an effort to swallow food, or to resist its administration—may then easily occur. In other cases a fatal attack of cardiac angina occurs without obvious premonitory symptoms. Paralysis may affect the diaphragm or intercostal muscles, in either case imperilling life not only directly, but indirectly by favoring the occurrence of broncho-pneumonia. If the patient is at rest in bed paralysis of the diaphragm produces no symptoms, but may be recognized by the inversion of the normal movements of the epigastrium in respiration. When it exists, however, slight exertion causes severe dyspnœa, and if the intercostal muscles be weakened also, death may suddenly be brought about.

The prognosis of diphtherial palsy is on the whole good. Recovery is the rule, except in cases in which there is distinct disturbance of the respiratory or cardiac functions, and all paralysis has passed away in a few weeks, or at most a month or two. If paralysis of the pharynx does not extend after two or three days, there is good reason to believe that it will remain limited, and the chief danger to be guarded against is the entry of food into the larynx. On the other hand palsy of the cervical muscles or marked ataxy calls for the greatest care, and a guarded prognosis, since respiratory or cardiac palsy ensues in many cases of this type.

The **diagnosis** of diphtheria is often difficult and uncertain. Mistakes arise most often in cases with insidious onset, in which there are no symptoms to call special attention to the throat; hence it is a sound rule to make an examination of the fauces a matter of routine in all cases. When the fauces and pharynx are the parts affected by diphtheria, diagnosis is, as a rule, relatively easy if a thorough in-

spection be carried out. Distinct false membrane on the pillars of the fauces or uvula will always raise a strong suspicion and warrant the immediate isolation of the patient. The same is true of well-marked false membrane on the tonsils. Acute follicular tonsillitis, which is occasionally met with even in infancy, may, if attended by a coherent muco-purulent exudation from the crypts, recall diphtheria, beginning at several different points on the tonsils; and in some few cases discrete tonsillitis, both acute and sub-acute, is really diphtherial. Ulceration of the tonsils, usually secondary to ulcerative stomatitis, is sometimes accompanied by ulceration of the soft palate; the ulcer is usually shallow, and its surface is covered by a yellow muco-purulent exudation presenting little resemblance to the yellowish white, firm, diphtherial membrane. Bacteriological examination has shown that pharyngitis, apparently simple, is, in certain rare instances, really diphtherial. The occurrence of such cases lends support to the opinion that the safest rule to follow is that adopted by many who have had large experience of schools—to regard all cases of sore throat as infectious until the contrary has been proved.

In coming to a decision much assistance may be obtained from *bacteriological examination*. If the clinical signs render diphtheria probable, the detection of the diphtheria bacillus will clinch the diagnosis. Under the same circumstances, however, failure to detect the bacillus does not disprove the existence of diphtheria, especially if the examination be made late in the case. On the other hand, in the absence of characteristic clinical signs, the detection of the bacillus in the secretions of the throat or mouth does not warrant the clinical diagnosis of diphtheria, although it would render obligatory the antiseptic treatment of the mouth and throat, and the isolation of the individual from other children.

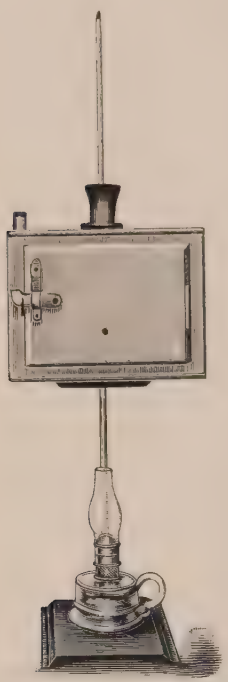
[Too much importance cannot be attached to the bacteriological investigation above mentioned. Provision for such investigation is now made in most of our American cities by their respective boards of health. The technique of the examination is simple, easily carried out and of great satisfaction and value to the attending physician. Those who have not access to a city laboratory will find the apparatus and method described by Koplik[1] of practical help in making a rapid diagnosis of diphtheria. It is briefly as follows: Wipe the throat with one of the swabs now in general use. Distribute the material thus obtained over the blood serum. Raise the temperature of the incubator—the small water oven in use in all chemical laboratories for drying purposes—to 38° C. by means of a Bunsen burner or alcohol lamp. (See cut.) Place the inoculated test-tube in the incubator and leave for two and one-half to three hours, keeping the

[1] *Trans. Amer. Ped. Soc.*, Vol. IX., 1897; *N. Y. Med. Journal*, August 1, 1896.

temperature at 38° C. At the end of this time remove the tube, scrape carefully the surface of the serum, prepare cover-glass in usual way, stain with blue of Loeffler, mount in Canada balsam, and examine with oil immersion lens. The whole method depends upon forcing the growth of the bacilli at 38° C., the temperature at which they thrive best.]

The **prognosis** should be guarded in all cases of diphtheria. The younger the child the greater the danger to life, especially through laryngeal obstruction. Cases which at the outset appear slight, may rapidly become very grave; and even in the mildest the possibility of subsequent palsy must be borne in mind. With regard to pharyngeal diphtheria, the prognosis is on the whole worse the greater the extent of membrane and the rapidity of its spread. Early enlargement of the lymphatic glands is also a bad sign, and indicates, probably, a mixed infection. Persistent vomiting and diarrhœa are also of evil augury, as is also a great diminution in the quantity of the urine, or the persistence of more than a trace of albumen. Irregularity of the pulse or failure in its strength indicate that the heart is becoming embarrassed, and greatly aggravate the prognosis. Nasal diphtheria, if accompanied by much sero-purulent discharge, is probably due to mixed infection, and the mortality of such cases is very high. In diphtheria of the larynx the prognosis is always grave, since to the ordinary risks of diphtheria there are superadded those of obstruction, and the special liability to diphtherial tracheitis and bronchitis, and to broncho-pneumonia. Progressive increase of dyspnœa, indicating growing obstruction, or continuous dyspnœa with blanching or cyanosis of the face and failing pulse, indicating the onset of broncho-pneumonia, warrants a very serious opinion as to the prospects of recovery.

FIG. 9.



Incubator employed in making a rapid diagnosis of diphtheria.

The introduction of **antitoxic serum** has modified materially the prognosis of diphtheria. The value of the remedy may be judged either by individual clinical experience, or by the statistical method. The latter presents great difficulties in arriving at an absolutely trustworthy conclusion, because diphtheria varies greatly in the severity of the toxæmia which it produces, in the danger connected with its local manifestations, and in the character of the epidemic. Further,

the age of the patient and the date at which treatment can be commenced, influence the result. Certain of the sources of error in forming a conclusion may be eliminated if the statistics deal with a sufficiently large number of cases. The statistics of the Metropolitan Asylums Board for 1894, the year before the introduction of antitoxin (3,042 cases); for 1895 and 1896, the first years in which it was generally but not exclusively used in the hospitals of the Board (3,529 cases and 4,175 respectively);[1] and the statistics contained in the report for 1895 of the American Pediatric Society, dealing with some 5,000 cases in private practice may be quoted. From the statistics of the Pediatric Society a certain proportion of the milder cases were eliminated, and some were moribund when treatment was commenced. On the whole it seems fair to conclude that the statistics from both sources are somewhat less favorable to antitoxin than the reality. The percentage mortality in the Metropolitan Asylums Board hospitals in 1894, without antitoxin was 29.6; in 1895, in all cases, those with and those without antitoxin, 22.5; in 1896, 20.8; the Pediatric Society, all cases treated with antitoxin, 12.3. Diphtheria is a much more fatal disease in children under five than at more advanced ages; in the Asylums Board hospitals the reduction of mortality in children under two years was from 61.9 to 48.5 in 1895, and 45.48 in 1896; in children from two to five, from 43.7 to 30.7 in 1895, and 26.9 in 1896. The Pediatric Society's statistics for the same ages give a percentage mortality of 23.3 and 14.7 respectively. The beneficial effects of antitoxin are seen most conspicuously in cases which come under treatment early in the disease, as is shown in the following table:—

Table showing the Day of Disease on which the Patient came under Treatment, and the Mortality per cent.

Day of Disease.	Metropolitan Asylums Board.			American Pediatric Society.
	Without Antitoxin.	All Cases With and Without Antitoxin.		With Antitoxin.
	1894.	1895.	1896.	1895.
First	22.5	11.7	4.7	4.9
Second	27.0	12.5	12.8	7.4
Third	29.4	22.0	17.7	8.8
Fourth	31.6	25.1	22.5	20.7
Fifth and over	30.8	27.1	24.6	35.3
Unknown	—	—	—	8.2

[1] Not all the cases in the Asylums Board hospitals were treated with antitoxin. Those which were moribund at the time of admission, and a large proportion of the milder cases (together numbering 1,347) were not so treated; in comparing the statistics it seems just therefore to take the whole series of cases for 1895 and 1896.

Table of 4,071 Bacterially-verified Cases of Diphtheria treated with antitoxin by the Chicago Health Department between October 5, 1895, and February 28, 1899; showing the Numbers first treated on Different Days of the Disease and Proportion (per cent.) of these to Totals Treated; Numbers of Deaths and Mortality (per cent.) of each day Group; Total Deaths and Mortality (per cent.) of the Totals of each Period and of the entire Period of 41 Months.

Periods.	Totals Treated.	By days (or Stages) of Disease when Cases were first Injected with Antitoxin.																				Total Deaths.	Mortality—Per cent.
		Numbers treated on—					Per cent. of Totals Treated on—					Deaths among those treated on—					Mortality per cent. of those treated on—						
		1st day.	2d day.	3d day.	4th day.	Later.	1st day.	2d day.	3d day.	4th day.	Later.	1st day.	2d day.	3d day.	4th day.	Later.	1st day.	2d day.	3d day.	4th day.	Later.		
Oct. 5, 1895, to Dec. 31, 1896.	2297	168	529	1044	382	174	7.3	23.0	45.4	16.7	7.6		8	29	57	57		1.51	2.77	14.9	32.7	151	6.57
Jan. 1 to Dec. 31, 1897.....	866	126	246	221	147	126	14.6	28.4	25.5	16.9	14.6	1	6	17	8	28	0.78	2.43	7.69	5.44	22.2	60	6.93
Jan. 1 to Dec. 31, 1898.....	709	54	206	182	141	126	7.6	29.0	25.7	19.9	17.8		3	8	12	29		1.45	4.39	8.51	23.0	52	7.33
Jan. 1 to Feb. 28, 1899.....	199	7	37	62	50	43	3.5	18.6	31.2	25.1	21.6			3	5	5			4.84	10.0	11.63	13	6.53
Totals and percentages.....	4071	355	1018	1509	720	469	8.72	25.00	37.07	17.69	11.52	1	17	57	82	119	0.28	1.67	3.77	11.39	25.37	276	6.77

Opinion founded on clinical observation is, almost without exception, favorable to the influence of antitoxin, if its use can be commenced during the first three days of the disease. It produces an amelioration of the general symptoms attended usually by a fall of temperature and return of appetite. It stops the spread of the membrane, and leads to separation of that already formed. Under its use the proportion of cases in which the larynx is affected secondarily is diminished, and when the larynx has already become affected it renders the case less severe, diminishes the danger of suffocation, and causes the results after intubation and tracheotomy to be better. In the Asylums Board hospitals the mortality after tracheotomy fell from 70.4 to 49.4 in 1895, and 41.0 in 1896; while the percentage of laryngeal cases in which tracheotomy became necessary fell from 56 to 45.3 in 1895, and 41.0 in 1896. The statistics of the American Pediatric Society show a mortality of 25.9 per cent., after intubation in cases treated with antitoxin, as against 51.6 per cent. after intubation combined with calomel fumigations which had previously given the best results.

[More recent statistics confirm still further the value of antitoxin and emphasize the importance of early administration. The report of the Chicago Board of Health for February, 1899, is of special interest and shows how life can be saved by antitoxin even under the most adverse circumstances. The records given below are based exclusively upon the results in cases of the city poor living in unhygienic surroundings and treated by members of the Board of Health, especially assigned to this work.

A study of the above table shows a record of 4,071 cases of true diphtheria treated during a period of 41 consecutive months, with a mortality rate of 6.77 per cent., and of 418 cases treated in 4 months with a mortality of 4.11 per cent.

The low mortality among cases treated on the first day, only 0.28 per cent., gradually increasing to 1.67 per cent., 3.77 per cent., 11.39 per cent., and 25.37 per cent. on the second, third, fourth, and later days respectively, shows the great importance of early administration.

The second table given below shows the result of treatment by ages and by day of disease for four consecutive months.

Nine per cent. of these cases, or 38 in number, required intubation, with 30 recoveries and 8 deaths, a mortality of 21 per cent.]

With regard to *the influence of antitoxic serum on complications*, it must be observed that the fact that a larger proportion of serious cases survive must tend to increase the proportion of complications. This is especially the case in regard to the nervous system, and the statistics at present available tend to show that the proportion of cases in which paralysis ensues is not diminished, if indeed it be not

Table of Results of Treatment, November, December, 1898–January, February, 1899: By Ages and Day of Disease.

Total Treated by Ages.				Day of Disease when first treated.	Recovered.				Died.			
Under 1 year.	1 to 5 years.	5 to 10 years.	Over 10 years.		Under 1 year.	1 to 5 years.	5 to 10 years.	Over 10 years.	Under 1 year.	1 to 5 years.	5 to 10 years.	Over 10 years.
2	16	7	6	1st day.	2	16	7	6				
9	48	20	21	2d day.	9	47	20	21				
7	53	39	15	3d day.	7	50	38	15		3		
7	43	37	13	4th day.	5	39	36	13	2	4	1	
	29	34	17	Later.		25	26	16		4	6	
25	189	137	72	Totals.	23	177	127	71	2	11	7	
								Mortality 8 %		5.82	5.25	

increased. Sevestre[1] believes that while early localized paralyses (palate) are not less frequent, cases of generalized paralysis are more rare. Injection of the serum is followed in a considerable proportion of cases by slight temporary albuminuria. On the other hand, there is no adequate ground for the assertion that the use of antitoxin is followed by an increase in the proportion of cases in which nephritis occurs as a complication. In some cases in which albuminuria exists before the injection, it diminishes rapidly; in others, it remains uninfluenced. In a large proportion, approaching one-half, the injection is followed by a rash. In some cases the rash appears on the third, fourth, or fifth day; it is then usually urticarial, and disappears in a few hours or a day or two at most. In other cases a rash comes out from the twelfth to the fourteenth day. This is sometimes very extensive, resembling the eruptions of scarlet fever, measles, or septicæmia. It is often accompanied by pyrexia which may persist for several days. Occasionally pyrexia occurs without rash. Joint pains, sometimes severe and aggravated by movement, but not accompanied by obvious effusion, and lasting only for a few days, occur in a small proportion of cases. Abscess at the site of injection is rare, and appears always to be due to some failure to secure asepsis.

Since the results are much better when the injections are made early, the first injection should be given as soon as the diagnosis is made. The needle, and all parts of the syringe, must be sterilized by placing them in cold water, which is then raised to the boiling-point, and the apparatus is boiled for five minutes, removed from the water with sterilized forceps, and placed to cool[2] on a piece of boracic lint, or a clean napkin. The serum should be quite clear, and if possible, recently prepared, though even when a year old its properties may be unimpaired. The injection is made into the sub-

[1] "Traité des Maladies de l'Enfance" (de Grancher, Comby, et Marfan), t. i., p. 640.
[2] The serum may be coagulated if the syringe is used while hot.

cutaneous tissue of the flank by picking up a fold of skin and thrusting the needle through the true skin. The skin is prepared by washing with soap and water, and bathing with sublimate 1 in 1,000,

FIG. 10.

Diphtherial infection of uvula and anterior pillars of fauces, showing the disappearance of the membrane after injection of antitoxic serum (A, 18 hours; B, 24 hours, and C, 36 hours after injection). (From drawings by DR. ROWLAND POLLOCK.)

after which it is covered with absorbent cotton. After the injection the pad of cotton-wool is replaced, and retained by a bandage. The syringe is cleansed with cold sterilized water. For children under

two years, and in mild cases over that age, the first dose should be 1,000 units; in severe cases above that age, 1,500 to 2,000. If there be no improvement, the dose should be repeated in from eighteen to twenty-four hours, or in bad cases even earlier, and a third dose may be given if necessary.

In those cases in which the serum produces its characteristic effect, the false membrane becomes in five or six hours whiter and more prominent, and is surrounded by a zone of deeply injected mucous membrane. The false membrane begins to become detached after about twenty-four hours or a little later, and is separated on the second or third day (Plate I.). After the injection of the antitoxic serum there is frequently a rise of temperature (1° to 3° F.); the maximum is reached in four or five hours, and a decline begins six to ten hours later, so that on the second day the temperature is normal. The pulse rate rises also, and often does not fall for several days. The effect on the general symptoms is parallel; there is at first some aggravation of the malaise, but after twelve to eighteen hours this begins to diminish, and at the end of twenty-four hours, if the injections have been given soon after the onset of the disease, the patient looks and feels much better. In cases of mixed infection the change in color is less marked, the separation of the membrane less early, and less complete, and the effect on the general condition of the patient slight or wanting.

The **general treatment** of diphtheria resolves itself into the attempt to conserve the strength of the patient. The disease is of an extremely exhausting and depressing character, and the first essential is rest. This cannot always be obtained by the same means, and every case must be considered for itself. As a general rule the patient should be kept in bed, and as much as possible in the recumbent attitude. The room in which the child is nursed should be large, well-ventilated, and have as little furniture as possible. In serious cases good nursing is very important. Food should be given in small quantities at frequent intervals, but this is in too many cases a counsel of perfection, and we must be content to give as much as the child will take without resistance or struggling or choking, which are to be avoided. It will often be necessary to supplement the food taken by the mouth by nutrient suppositories. Alcohol is generally well borne in cases in which there is much depression, but it must be given freely. Adynamia must be treated on the general principles stated in Chapter V.

In the treatment of diphtherial palsy the main indications are rest and careful feeding. Soft but not too liquid food is to be preferred, and it is a good plan to induce the child to take its food while lying face downwards. In extreme pharyngeal paralysis it may be necessary to administer liquid food through the nasal tube. Signs of

respiratory failure must be combated by hypodermic injections of strychnine, inhalations of oxygen, and absolute repose; of cardiac failure by camphor, ether, or other stimulant injections. It is the custom to give strychnine in all cases of diphtherial palsy, though in the milder, limited cases it is not necessary. Nourishing food, iron, cod-liver oil, are of use during convalescence, which is usually protracted, a condition of feeble health remaining usually for some months. Change of air will be of advantage if not taken too soon, and if the patient be guarded against fatigue. Prolonged rest for body and mind should be insisted upon; and, as a rule, a child should not be allowed to attend school until the knee-jerks have returned.

The **local treatment** of pharyngeal diphtheria which has been much followed, has had in view two objects—the destruction or disinfection of the false membranes, or the disinfection of the general cavity of the mouth and pharynx, with the hope of preventing decomposition and secondary infection. For the first purpose the list of caustic, astringent, or disinfectant drugs used is very long. To be effectual the solutions used must be strong, and must be applied accurately to the affected part. To do this the child must be completely under control, and the operator must be certain that he has all the surface covered by membrane under inspection. Practically, in young children these ends cannot be attained, and the attempt to make local applications excites terror and resistance, so that it becomes very difficult to ensure that the affected parts are completely and exclusively medicated. As Jacobi has well said, "There are cases which do not show the harm done. The fact is, that neither the galvano-cautery, nor carbolic acid, nor tannin and glycerin, nor perchloride or subsulphate of iron can be applied with leisure and accuracy to the very membrane alone, except in the case of very docile and patient children. In almost every case the surrounding epithelium is getting scratched off or injured," and thus the spread of the diphtherial membrane is favored. One of the best local applications is tincture of iodine, which penetrates the membrane while producing little surrounding irritation. Löffler's solution, which is an alcoholic solution of toluol, creolin, and menthol, has been highly praised.

[Menthol, 10 parts by weight.
Toluol, 36 parts by measure.
Creolin, 2 parts by measure.
Alcohol, to 100 " " "

Appendix.]

The most thoroughly local medication is that devised by Gaucher, but it is extremely painful.

[Camphor, 20 parts.
Carbolic Acid (Crystals), 5 "
Tartaric Acid, 1 "
Castor Oil, 15 "
Alcohol (90 degrees), 10 "
Dissolve the carbolic acid in the alcohol, add the camphor, then the tartaric acid, and lastly the castor oil. Appendix.]

A large number of small absorbent cotton swabs are prepared; several of these are used in succession to remove the false membrane as far as possible; next a strong solution of camphor and carbolic acid in spirit and castor oil is applied very thoroughly to the denuded surfaces; and, finally, after an interval of ten minutes, the throat is washed out by a stream from an irrigator so regulated as to be strong enough to excite forcible contraction of the pharynx, and thus prevent deglutition. About three pints of liquid, which may be simply boiled water, or carbolic acid 1 in 100 should be used. The process must be repeated every three or four hours. It is extremely trying to adults, and obviously inapplicable to young children. Short of this, however, it is very doubtful whether local applications with a brush or swab will effect any more than the use of sprays and douches, which can be much more easily applied. They assist in the removal of shreds of membrane, check decomposition in the mouth, and when rendered astringent, diminish the tendency to catarrh. For this purpose solutions of carbolic acid 1 in 100, of salicylic acid 1 or 2 in 1,000, of boric acid (saturated), of perchloride of mercury 1 in 5,000, or of potassium permanganate 1 or 2 in 1,000 may be used. The main point is to use a large quantity of the solution, and to see that the stream is sufficiently strong to excite reflex contraction of the pharynx, so that most of the solution is returned through the mouth. The child must be held firmly by an assistant, with the head bent forward, and the mouth be opened sufficiently, and the tongue so far depressed as to ensure to the solution free access to the pharynx and egress from the mouth. For nasal diphtheria, antiseptic solutions must be injected into the nostrils, or where this is difficult, owing to the age of the child, the spray may be used. The nozzle of the syringe should be covered by a piece of india-rubber tubing and the injection thrown directly backward. As much of the fluid injected is swallowed, it is not advisable to use poisonous drugs, such as perchloride of mercury.

In laryngeal diphtheria local applications are, practically, out of the question in children, but much relief may be afforded by keeping the child constantly in moist, warm atmosphere (steam). A steam tent should be used, but if some relief is not obtained in a few hours it is not, as a rule, desirable to persevere. [Where the symptoms of laryngeal diphtheria are so severe as to require surgical interference, the operation of election is now universally conceded to

be intubation, tracheotomy being reserved for those cases in which intubation has been tried without success.]

Of the multitude of drugs recommended for internal administration the best are the tincture or solution of perchloride of iron in pharyngeal diphtheria, and perchloride of mercury when the larynx is affected. In either case the drug must be given in small doses frequently repeated, the iron salt every half-hour or hour, in such doses that an infant takes ʒj to ʒiss, during the twenty-four hours, the mercurial hourly, so that gr. $\frac{1}{4}$ is taken during the same period. The dose should be reduced after a few days. Children of three years will take twice this quantity.

[*Immunization.*—There can to-day be no doubt of the value of immunizing doses of antitoxin given to those exposed to diphtheria, and all such should receive at the earliest possible moment an amount of the serum varying with the age of the individual from 50 to 500 units. An infant under three months should receive 50 units; from five to ten years the dose is 200–400 units; above ten, 500 units. The length of time for which immunity will be conferred by these doses is variously stated at from ten to twenty days.]

CHAPTER IX.

MALARIAL FEVER.

The Hæmatozoon—Varieties of Malarial Fever: Quotidian; Æstivo-autumnal; Pernicious—Malarial Cachexia—Diagnosis—Prognosis—Treatment.

Malarial fever is due to infection by the hæmatozoon first described by Laveran in 1880. Different clinical types of malarial fever correspond to certain morphological peculiarities of the associated parasite, which may perhaps indicate specific differences. Two main varieties may be distinguished: (1) the parasite of simple intermittent fever (*a*) tertian, (*b*) quartan; and (2) the parasite of irregular, grave (æstivo-autumnal) fever. While in the human body the hæmatozoon is in all varieties and at all stages of its career, except that of free spore, an intra-corpuscular parasite. The tertian and quartan parasites pass through a series of changes in the red blood-corpuscles, ending in a process of segmentation and the formation of spores, which, on being set free, invade a fresh set of corpuscles, and the cycle commences again. In the case of the tertian parasite the cycle occupies forty-eight hours, of the quartan seventy-two hours. A quotidian fever, that form most often seen in children, is produced by a double tertian infection, segmentation taking place on different days; or, more rarely, by a treble quartan infection. The characteristic paroxysm of malarial fever corresponds with the breaking up of the parasite and the escape of the spores into the liquor sanguinis on the completion of the cycle by the process of segmentation. The development of the parasite of the æstivo-autumnal fever, though often approximately tertian, is less regular, being sometimes apparently quotidian. Segmentation takes place, probably, in the spleen, bone-marrow, brain, and viscera, rarely in the peripheral blood. The fever is often irregular, the paroxysms sometimes imperfectly marked, and the remissions often incomplete. The symptoms produced by malarial infection must be attributed, in part, to the actual destruction of the infected corpuscles, and in part (fever, etc.) to toxins which are assumed to be liberated in the blood in the breaking up of the parasite after segmentation.

Opinions differ as to whether infants and children are more or less liable to suffer from malaria than adults, the truth being, in all probability, that the liability is the same. Malaria may occur at

any age, and has been observed so soon after birth (eighteen hours) that the infection must have been intra-uterine.

In children over the age of about six years, the symptoms of malarial infection present no characters to distinguish them from those observed in adults, but in infants and young children the paroxysms are less regular, the mode of onset more insidious, and the risk to life greater. The attacks are usually quotidian, but the paroxysms are not so well marked, and the remissions are less complete than in a typical case in an adult. The cold stage is not usually marked by definite rigors, but the infant vomits, becomes blue about the lips and hands, and is peevish or strikingly somnolent. Occasionally, convulsions are the first symptom, but frequently the symptoms of the first stage are so little marked that the infant is not noticed to be ill until the febrile stage begins. When this is established the face is flushed, the surface pungently hot, and the temperature 104°, 105°, 106° F., or even higher; after two to eight hours the febrile stage ends, and the temperature falls considerably, but not always to normal; profuse perspiration, usually observed in adults at this stage, is seldom or never observed in infants and young children. The spleen, after one or two attacks, will be found to be enlarged in nearly all cases. In those cases in which the onset is insidious, the patient becomes languid and peevish, loses appetite, complains of abdominal discomfort, and there is often diarrhœa. The spleen and liver will be found to be enlarged, and the skin has an earthy yellow tint. Some continuous pyrexia is present with, perhaps, irregular fluctuations; after a time the exacerbations grow more severe, the remissions more pronounced, and a quotidian type of fever becomes distinguishable. In such cases, gastro-intestinal disturbance of various kinds, but especially obstinate diarrhœa with mucous stools, is common. Unless suitable treatment be adopted at an early date malarial infection quickly produces a profound deterioration in the child's health, and the patient becomes emaciated, sallow, and extremely anæmic. In such a condition, it may easily succumb to some intercurrent infection.

The *pernicious* forms of malarial fever appear to be comparatively rare in children. Occasionally, however, infants after one or two paroxysms, suddenly pass into a comatose condition with high temperature or hyperpyrexia; in older children, eclampsia during the febrile stage marks a severe form of infection.

Repeated attacks of malarial fever, or prolonged residence in a malarial district, even without distinct attacks, may lead to the development of a condition of *malarial cachexia* characterized by wasting, anæmia, and enlargement of the spleen, which may attain an immense size. The patients suffer also from intestinal catarrh, œdema of the extremities, petechiæ, and epistaxis. In children who

have suffered from ague various neuralgic pains may occur and lead to errors in diagnosis unless their malarial origin be recognized. Frontal headache is a common symptom, and if associated with drowsiness, vomiting, and constipation, as is sometimes the case, may lead to a suspicion of tuberculous meningitis.

The *diagnosis* of malarial fever may be made by a recognition of the hæmatozoon in the blood; apart from this it must depend upon a history of residence in a malarious district, the periodicity of the symptoms, the enlargement of the spleen, the effect of quinine, and upon the exclusion of other conditions which could account for the fever or other symptoms. If the blood be not examined, it may be very uncertain, and there can be no doubt that in countries in which malaria prevails, very many cases are attributed to its influence upon very slender grounds. The attempt to distinguish the severer forms from typhoid fever was, at one time, in many districts altogether abandoned; the examination of the blood for the malarial parasite, and the serum test for typhoid fever will set many doubts of this kind at rest. Further, the temperature curve of malarial fevers during the first week is never so regular as that of typhoid fever, and the exanthem does not occur. From tuberculosis there may be, as already said, temporary hesitation in distinguishing the more irregular forms of malaria, but the effects of treatment will generally set such doubts at rest, and the same remark applies to the irregular fever of pyæmia, in which, moreover, some initial lesion will usually be discoverable.

The prognosis is good except in those cases in which the symptoms develop with great rapidity and the patient quickly becomes comatose.

The treatment of malaria consists in the main of the proper administration of quinine; it has the effect of causing all forms of the parasite, with the exception of the crescent body, to disappear from the blood. Given some hours before a paroxysm it will stop not that paroxysm but the next. In young children, in whom the periodicity is not often well marked, it is perhaps best to give the amount considered appropriate in divided doses during the twenty-four hours. Except that the bitterness of the drug leads them to dislike taking it, infants and children tolerate quinine well, and in ordinary cases an infant may be given at once gr. ss to gr. j, three times a day, increased rapidly if the desired effect is not produced; in mild cases, however, as good an effect will be obtained with the smaller dose. It may be given in solution with syrup of orange, or if there is much repugnance to it, in powder suspended in a teaspoonful of milk. Vomiting is sometimes exceedingly troublesome, and quinine may be given by the rectum, either by enema to which a drop of tincture of opium is added, or better, in a cocoa-butter suppository; the dose should be double that given by the mouth. If other expedi-

ents fail, it may become necessary to give the drug by hypodermic injection. In the pernicious forms in which the patient is comatose, and in which it is important to produce the effect as rapidly as possible, the same method must be resorted to from the first. For hypodermic injection the best salt is the hydrochlorate or the hydrobromate. The injection (gr. ss to gr. j), should be made deeply in the buttocks or back.

Malarial cachexia, if it have not reached too advanced a stage, will usually improve under the administration of iron and arsenic, careful dieting, and removal to a non-malarial district.

[Malarial fevers occur in this country in southern New England, along the Atlantic Coast south of New York, in the Gulf States and in the Mississippi Valley; in the last-named region severe forms are met with. Mild forms of the disease also occur about the Great Lakes, in the Middle States and on the Pacific Coast.

Thayer thinks that the more irregular course of malaria in children is probably due to "infection with multiple groups of the parasites or to the lack of arrangement of the parasites in well-marked large groups." He also thinks that the chronic malarial cachexia so common in young children and infants is due to failure to recognize the disease and consequently improper treatment.

Examination of the blood for the parasite should always be made in the obscure irregular fevers of early life.]

CHAPTER X.

TUBERCULOSIS: ETIOLOGY; PATHOLOGY.

The Tubercle Bacillus—The Tuberculous Diathesis—Sources of Infection—Milk—Predisposing Diseases—Sites of Primary Infection: Naso-Pharynx and Cervical Glands; Ear; Intestines—Varieties of the Tuberculous Process—Age Incidence—Prevalence of Tuberculosis in Childhood—Sex.

Etiology.—Two factors have to be considered, the susceptibility of the individual and the opportunities for infection. The importance of the former has been alternately exaggerated and minimized. While it is certain that tuberculosis cannot occur in the absence of the specific infective agent—the *tubercle bacillus*—it is equally certain that under favorable circumstances the human organism, whether that of the child or of the adult, is able continuously to destroy tubercle bacilli which enter the lungs or intestines in small numbers. This must occur with great frequency in all populous places.

A circumstance which has much influence in increasing the susceptibility of the individual is the inheritance of a special type of constitution, the so-called *tuberculous diathesis*. The main causes diminishing the power of resistance are over-crowding, insanitary surroundings generally, and malnutrition. Deficient ventilation of living rooms has a double action, since it tends to deteriorate the general health, while at the same time it increases the chances of aërial infection.

The main *sources of infection*, in the child as in the adult, are through the air and through food. The risk of infection of the lungs and air passages by the tubercle bacillus disseminated through the air by the pulverization of dried sputum, is well known and need not be discussed here. Whether children are more or less liable than adults to infection in this way may be left an open question, but there is strong reason to believe that they are more liable to infection, or, at least, more often infected through food, owing either to a lower power of resistance or, more probably, to the fact that one of their main articles of food—milk—is specially liable to carry the infection. Tuberculosis is a very common disease of milch cows, but their milk only becomes infective when the udder is the seat of tuberculous disease. According to Sidney Martin[1] "the milk of

[1] "Report of the Royal Commission on Tuberculosis," part iii. (1895), p. 39.

cows with tuberculosis of the udder possesses a virulence which can only be described as extraordinary," and is unfit for human consumption. Woodhead[1] has pointed out that such milk, when added even in small quantities to milk from a healthy source, can impart to it infectious qualities. In exceptional cases tuberculosis is truly congenital, the infection having taken place during intra-uterine life. In a few cases infection has taken place through the skin, as in the rite of circumcision, or from saliva used for the lubrication of earrings, or for mixing the paint for tattooing. Possibly the habit which most infants and young children have of putting every object which they pick up into their mouths may occasionally be the mode of infection.

Among *predisposing* causes mention must be made of certain diseases. Of the acute diseases, the most important in this connection are measles and whooping-cough; of the chronic, catarrh of the respiratory and gastro-intestinal mucous membranes. As in the adult, catarrhal affections of the bronchi and lungs are often forerunners of tuberculosis. The epithelial degeneration thus produced doubtless favors the development of the specific infection. It is owing, probably, to their proneness to produce catarrh and lymphadenitis that measles and whooping-cough so frequently determine the onset of tuberculosis; but in other cases they act by rousing into activity glandular tuberculosis, already in existence, but in an absolescent or latent stage (Geill).

There is good reason to believe that in children infection takes place, in some cases, through the *nasopharynx*. It would appear that in them the tubercle bacilli can be carried from the lymphoid tissue of the tonsil and pharynx to the cervical lymphatic glands, even though there be no obvious tuberculous lesion of the mucous membrane. To this mode of infection is probably due the chronic tuberculous adenitis of the neck (strumous glands) so common in early life. The after course in these cases varies greatly. In some, probably in the majority, the disease does not become generalized. In others, general tuberculosis ensues, and has been known to follow the operation of scraping out the enlarged glands. In others again, the infection descends from gland to gland, until it reaches the lymphatic glands at the root of the lung, or the pleura near the apex, or both. From these situations the lungs become involved by extension. Woodhead, arguing from the result of feeding experiments,[2] expresses his belief that this method of infection of the

[1] *Ibid.*, p. 149.

[2] Especially in the pig, which in its omnivorous diet, if not, as the pre-Vesalian anatomists affected to believe, in structure also, is "likeliest the human form divine." He says, referring to this animal (*Lancet*, 1894, vol. ii., p. 958): "In many of these cases the process can be traced from the glands in the tonsil down into the neck, and so on to the thorax by the mediastinal and post-sternal glands, and by the intercostal

glands of the neck through the tonsils must be of comparatively frequent occurrence, especially in children living under insanitary conditions and subjected to various devitalizing influences.

In this connection it may be pointed out that tuberculous disease of the *ear* is probably by no means uncommon in children, and especially in infants, Körner[1] states that frequently at the *post-mortem* examination of children who have died of general tuberculosis, there is found, along with suppuration of the tympanic cavity and caries, or necrosis of the temporal bone, tuberculous menigitis, or tubercle in the substance of the brain, and points out that Henoch lays stress on the frequency of the combination of caries of the petrous portion with intracranial tuberculosis. In some cases the tuberculosis of the ear is only a part of a widespread tuberculosis, but in others it appears to be the starting-point.

Infection by tuberculous milk may take place also through the *intestines.* As in the case of the pharyngeal lymphoid tissue, the small wandering cells of the lymphoid patches of the intestines take up the tubercle bacilli; under favorable circumstances, that is to say, in a vigorous individual, or when the number of bacilli is small, they are destroyed by the cells, but if they escape destruction one of two things may happen: they may develop in the lymphoid patches, producing tuberculous ulceration; or they may be carried by the wandering cells to the mesenteric glands, leading to an enlargement and finally to caseation of these glands (tabes mesenterica). It is not always the nearest glands which become infected. The bacilli succeed in running the gauntlet of the first chain. The bronchial glands, and finally the lungs, may become infected secondarily. Thus Woodhead writes,[2] "I have seen in case after case in children, and in animals fed on tuberculous material, the lungs markedly affected; but in a large proportion of these cases it has been possible to trace the course of invasion back from a caseous or old calcareous mesenteric gland, through the chain of retro-peritoneal glands, up through the diaphragm to the posterior mediastinal and bronchial glands, and so on to the lung. I have not seen this in a few cases only, but in dozens of children, in a few adults, and in many animals." The same observer believes that in infants primary infection of the lungs may take place occasionally by the direct entrance of infective tuberculous material derived from milk into the respiratory passages.[3] Tuberculous infection of the alimentary canal by meat contaminated with tuberculous material, which has been shown to be a

lymphatics and glands, and it is interesting in such cases to note how the lungs may be perfectly healthy, until the glands at their root, or in the pleura, have become distinctly affected."

[1] *Die otit. Erkrank. d. Hirns,* etc., 1896.

[2] *Lancet, loc. cit.,* p. 960.

[3] "Royal Commission on Tuberculosis," 1895, part iii.

very rare event at any age, must be extremely infrequent in infancy, and probably not much more common in childhood. It has been supposed that the practice of giving grated raw meat to infants might be responsible for producing intestinal infection, but this can very rarely occur even when beef is used for this purpose, and the danger may be obviated by employing mutton in its place.

Pathology.—It is not necessary in this work to describe the characters of the bacillus tuberculosis, nor the minute anatomy of the lesions to which it gives rise, since these subjects are now dealt with fully in all the text-books of medicine. In childhood, tuberculosis is more prone to be generalized from the first, or to become generalized at an early stage of a local infection, and the distribution of tuberculous lesions in childhood differs from that usually met with at adult ages. The lymphatic glands, the bones, and the meninges are affected more often during the first ten years of life than at any subsequent age. The severity and acuteness of tuberculosis varies greatly; this variation depends upon the degree of susceptibility of the individual, the mode of infection, and probably upon differences in the virulence of the bacillus from different sources.[1] Thus we have at one end of the scale acute tuberculosis, and at the other lupus, with many intervening types, of which scrofulous disease of bones and glands is the most definite.

Age Incidence.—*Congenital tuberculosis* has been observed, but it is so extremely rare that Virchow has never met with an instance. Straus[2] states that, in spite of the great amount of attention directed to the question, the opinion expressed by Cohnheim in the following words still remains true: "The number of indisputable cases of congenital tuberculosis is extremely limited, and can be counted on the fingers." In all well-authenticated cases the mother has been tuberculous.

Tuberculosis is rare under the age of three months, and is not often met with under six months; it then becomes more common, and is extremely fatal in the *second year of life*. Hutinel found that 3.5 per cent. of the infants under one year who died[3] in the Hôpital des Enfants-Malades were tuberculous. "Very different," he adds, "are the proportions after the first year; at the Hospice des Enfants Assistés a third of the children between one and two years present tuberculous lesions." Statistics from Kiel and Munich appear to show that, so far as a conclusion can be drawn solely from *post-mortem* observation, the liability to tuberculosis increases very rapidly

[1] The *Report of the Medical Officer of the Local Government Record* for 1888 contains a report by Dr. Lingard on the relations of scrofula, lupus, and tuberculosis, which affords experimental evidence on this point.

[2] "La Tuberculose et son Bacille." Paris, 1895, p. 530.

[3] During 1890, Straus, *loc. cit.*, p. 532. The Kiel and Munich statistics are quoted from the same author.

in the second year of life, and then progressively. The figures taken together are as follows :

Age.	Number of Post-mortems.	Tuberculous.	Percentage Tuberculous.
0– 1	1,487	67	4.5
1– 5	1,053	303	29.8
5–10	333	117	35.1
10–15	236	91	38.6

These figures, however, do not indicate the relative importance of tuberculosis as a cause of death at various ages, but rather the frequency of tuberculous lesions, from many of which recovery might have been possible under more favorable conditions of general health. It is important to form an opinion as to the relative importance of tuberculosis as a cause of death at various ages, because such knowledge will be of use in considering the subject of prophylaxis, and also in diagnosis, when, as is often the case, the signs and symptoms do not at once warrant a confident expression of opinion. The best statistics in this connection are probably those of Holsti, for the town of Helsingfors and its suburbs.[1] The number of deaths from tuberculosis during the years 1882–89 was 1,771. The following table shows the age-distribution, and also the proportion to 10,000 persons living at each age :

Age.	Number of Deaths.	Mortality per 10,000 Living.
0– 1	215	285
1– 2	180	260
2– 5	129	63
6–10	42	17
11–15	20	8
16–20	61	17
21–25	156	33
26–30	195	41
31–40	395	50
41–50	212	45
51–60	108	40
61–70	46	36
Over 70	12	20

The table brings out very clearly the extreme liability to tuberculosis during the first two years of life, and, since children under six months are very little subject to the disease, it indicates a rapid increase during the second six months of life.

With regard to *pulmonary tuberculosis*, Bertillon's statistics for Paris show that the mortality from the disease is very considerable

[1] *Zeitschr. f. klin. Med.*, 1893, Bd. xxii., s. 317.

under five years, falls to the minimum between five and ten years, and then begins to rise, reaching the maximum between the ages of thirty and forty-five. Würzburg's statistics for Prussia show very similar fluctuations in childhood and early adult life. Those of the Registrar-General for England and Wales show a very high rate of mortality from tuberculosis under five, a much lower rate from five to fifteen, and then a steady rise due to the increasing mortality from pulmonary consumption.

As Holsti has well observed, the great mortality under two years of age is an indication not so much of a greater prevalence of tuberculous disease at that age as of the fact that in infancy and early childhood it prevails usually in a form and with a localization which quickly cause death. Of 395 fatal cases of tuberculosis in the first two years of life, 265, or 67 per cent., were attributed to tuberculous meningitis, 81, or 20.5 per cent., to phthisis, 46, or 11.6 per cent., to tuberculosis or general tuberculosis, and 3, or 0.8 per cent., to intestinal tuberculosis. Holsti's observation on these figures—that while some cases of meningitis supposed to have been tuberculous may, in reality, have been simple, and some cases supposed to be general tuberculosis may have been typhoid fever, yet many cases really tuberculous were probably attributed to other causes, especially instances of tuberculous enteritis—is probably correct.

Prevalence of Tuberculosis in Childhood.—It is difficult to speak with confidence as to the extent to which tuberculosis prevails in children, for the statistics already quoted have dealt mainly with those who succumb to the disease, whereas there can be little doubt that many children suffer infection, but recover. Geill[1] has advanced some valuable evidence upon the subject, founded on the *post-mortem* appearances in a large number of children (at all ages under fifteen years) who succumbed to acute infectious diseases. His inquiry extended to 584 cases. Almost without exception, these children had been in good health—that is to say, they had presented no symptoms of scrofulosis before the onset of the acute disease—and in most there was before death no evidence that they were the subjects of tuberculosis in any form. *Post-mortem* 198, or 33.9 per cent., presented unmistakable naked-eye evidence of tuberculosis of one or more organs; 384 of the children were under two years of age, and of this number 60, or 21.2 per cent. were affected, while of those above two years between 46 and 47 per cent. presented tuberculous lesions. The existence of the tuberculosis no doubt, in some cases, determined death, and in others, by its depressing effect on the health, contributed to bring about that result; but, even so, the statistics afford evidence that a very large proportion of children, who appear to be in good health, have in reality become infected. In the majority,

[1] *Jahrb. f. Kinderhlkde.*, Bd. xxxii., s. 165.

probably, the infection does not spread beyond the lymphatic glands, which were affected in every one of Geill's cases.

Sex.—The statements as to the relative liability of the two sexes vary. The truth appears to be that, under two years of age, there is a slight preponderance of males; shortly before and after puberty, a preponderance of females.

CHAPTER XI.

CLINICAL VARIETIES OF TUBERCULOSIS.

Tuberculosis and Scrofula—General Tuberculosis: Acute and Chronic—Tuberculosis of Bones and Joints—Tuberculosis of Lymphatic Glands; the Cervical and Tracheo-bronchial Glands.

Clinical Varieties of Tuberculosis.—Tuberculosis in childhood may be acute, and then in a large proportion of cases is either a general disease from the first, or becomes generalized before the termination of the case; or it may be chronic, and may then be general or localized. When at first chronic and localized it is apt—owing to accident, surgical interference, or some intercurrent acute disease, as, for instance, measles—to become generalized and acute.

It is usual, and perhaps useful, to distinguish two types of constitution which are liable to suffer from tuberculosis in different ways. They have been described by Sir William Jenner in the following contrasted pictures:—*Tuberculosis.* "Nervous system highly developed; mind and body active; figure slim; adipose tissue small in quantity; organization generally delicate; skin thin; complexion clear; superficial veins distinct; blush ready; eyes bright, pupils large; eyelashes long; hair silken; face oval, good-looking; ends of long bones small, shafts thin and rigid; limbs straight. Children the subjects of tuberculosis usually cut their teeth, run alone, and talk early."—*Scrofulosis.* "Temperament phlegmatic; mind and body lethargic; figure heavy; skin thick and opaque; complexion dull, pasty looking; upper lip and alæ of nose thick; nostrils expanded; face plain; lymphatic glands perceptible to touch; abdomen full; ends of the long bones rather large, shafts thick." The first type furnishes perhaps the larger number of cases of miliary tuberculosis, of acute tuberculosis of the lungs, and of meningitis. The pathological characteristic of the scrofulous type is the proneness to inflammation of the skin and mucous membranes. Such children are peculiarly liable to various chronic forms of dermatitis, and of chronic ophthalmia, to chronic rhinitis with excoriation of the surface of the upper lip and thickening of its substance, and, above all, to chronic pharyngitis, and tonsillitis with consecutive enlargement of the cervical glands. To this type belong, probably, the larger number of cases of tuberculous, so-called "strumous," disease of bones

and joints. But the "tuberculous" type is by no means exempt from "scrofulous" disease of bones and glands, nor the scrofulous type from acute tuberculosis, whether general or limited to the meninges or lungs.

General Tuberculosis.—General tuberculosis occurs in children under two forms: (1) acute general tuberculosis, which conforms to the type of an acute specific fever; (2) chronic or subacute general tuberculosis, in which the patient passes into a cachectic condition.

Acute General Tuberculosis is due to a general infection involving many organs without a preponderating affection of any one. Infection of the general system is no doubt derived from some pre-existing local tuberculous lesion, but this may not have been observed during life, and after death may be recognized with difficulty. The primary lesion is most often in the tracheal, bronchial, or mesenteric lymphatic glands; more rarely in the bones, the lungs, or the kidneys. General infection is in many cases, probably in the large

FIG. 11.

i ii

i. Section of wall of small artery (intracranial) showing an early stage of tuberculous lesion. *a*, epithelium, unchanged; *b*, sub-epithelial layer proliferating, with one multi-nucleated giant-cell; *c*, elastic membrane; and *d*, media, unchanged. (After Hektoen, *Journ. of Exper. Med.*, vol. i.)

ii. Section of small artery (intracranial), including both walls, showing a later stage. A caseous focus, *f*, has perforated the elastic membrane, *c*, and has infiltrated the media and adventitia so as to form an aneurysmal dilatation of the vessel. Proliferation is commencing in the sub-epithelial layer, *b*, on the opposite side of the vessel. (After HEKTOEN.)

majority, brought about either by rupture of a tuberculous collection into a vein, or by tuberculous infection of the wall of a blood-vessel, leading in either case to the discharge of active infective material into the blood stream (Fig. 11, i and ii). General infection may also be produced by tuberculous disease of the thoracic duct. In some instances the determining cause appears to be an attack of

measles, whooping-cough, typhoid fever, or, more rarely, other infectious disease.

The symptoms commonly resemble those of typhoid fever. After a short period of failing health and loss of appetite, the increasing weakness, signs of fever, and perhaps some wandering at night, lead to medical assistance being sought. The patient is found to be apathetic, the face flushed, and the tongue small, red, or irregularly coated, and tremulous. The general aspect is one of severe illness, for which physical signs do not afford sufficient explanation. The pulse becomes rapid and feeble, the tongue dry, the lips cracked, and the face more flushed, while delirium is continuous, though worse at night. The course of the temperature may afford assistance in diagnosis owing to the fact that it seldom or never shows the regular rise and diurnal variations characteristic of typhoid fever. The rule is to find an evening rise to 102°–104° F., and a marked remission in the morning, but the temperature may reach the highest point at almost any period during the twenty-four hours, and the remissions may be so great that it may fall to the normal or below, and so remain for several days without any definite amelioration in the other symptoms; thus the pulse may be rapid and the general aspect and condition such as to lead to the expectation that the temperature is elevated, whereas the thermometer may show that it is little, if at all, above the normal. Further, throughout the whole course of the case, even for three or four weeks, there may be no rise of temperature, or only a transient elevation. Reinhold observed this in nine out of fifty-two cases. The respiration is usually hurried, especially in the early stages, and there is often some cyanosis, though physical examination will probably reveal, at most, signs of general bronchitis of slight intensity. In the latest stage the respiration may be of the Cheyne-Stokes type. Delirium is seldom noisy, and the child becomes more and more dull, until torpidity develops into coma. If intracranial infection ensue the delirium may become more active, and the hydrocephalic cry may be heard. (See "Tuberculous Meningitis.") Constipation is the rule, but diarrhœa may occur, and the stools may be blood-stained, though this is unusual. Enlargement of the spleen does not occur so early as in typhoid fever, but it may exist when the case comes first under treatment, and may eventually be considerable. The urine may contain a trace of albumen, and may give the diazo-reaction. There is no characteristic rash, but a few scattered red spots may appear (not in crops); they are irregular in size and often contain at the centre a swollen hair-follicle. Sometimes a very similar appearance is produced by flea-bites, and petechiæ, which may be due in part to the same parasite, are often numerous, especially about the wrists.

The diagnosis from typhoid fever, as will have been inferred from

what has been already said, is often extremely difficult. The possible relation of the case to others of typhoid fever should be inquired into. The observation of choroid tubercle or the discovery of tubercle bacilli in the blood where they have been demonstrated in a few instances, will, of course, set the question at rest. The application of the serum test for typhoid fever will afford valuable negative evidence. Less well-defined criteria are the absence of the characteristic rash, the irregular character of the temperature curve, and the nature of the stools if diarrhœa exist. It is often necessary to give a very guarded diagnosis at first, and it should be borne in mind that tuberculosis may occur as a complication or sequel of typhoid fever. Some cases of acute or subacute enteritis may resemble acute tuberculosis very closely. In such cases the child looks very ill, and may be delirious; it loses flesh rapidly, refuses food, and is very thirsty. The tongue, furred at first, becomes red and irritable. The abdomen is shallow, not very tender, and there may be no diarrhœa, or the stools, though frequent, are scanty. There is some irregular pyrexia, rising to a maximum of 102°–104° F. A guarded opinion must often be given at first, though the history of sudden onset, which is the rule in enteritis, the absence as a rule of bronchitis, of enlargement of the spleen, of irregularity of the pulse, and, it may be, the occurrence of other similar cases in the same house or neighborhood may render it possible to make a provisional diagnosis, even on the first occasion on which the patient is seen.

The prognosis is hopeless when the diagnosis is certain, and **treatment** produces no effect upon the course of the disease. The most that can be aimed at is to relieve symptoms and promote euthanasia.

Chronic General Tuberculosis occurs in infancy, and probably never after the second year. There is usually a history of an antecedent attack of bronchitis or broncho-pneumonia, or of measles or whooping-cough. The patient has apparently recovered from the acute attack, but does not regain his former health. In other instances the onset is insidious without obvious determining cause. In either case the characteristic symptoms are progressive emaciation, though the appetite is retained and is, indeed, commonly ravenous. In spite of the eagerness with which it takes food the infant is extremely thin, the skin, which hangs in loose folds on the limbs, is inelastic, and when pinched up returns only slowly to its original position. The face is pinched, but in young infants the sucking pads are usually prominent, the eyes are sunken, and the expression is tired, peevish, and anxious. The patient often has long eyelashes and an unusual amount of hair over the back and other parts. The liver and spleen are both enlarged, and there are small shotty glands in the axillæ and groins. The temperature is not raised, or only to

a slight degree and at irregular intervals. Not infrequently it is subnormal. There may be no physical signs of any pulmonary disorder, though in some cases there is some evidence of bronchitis, or of consolidation at the apex, hilum, or base. A dry cough is the rule, and an intercurrent attack of bronchitis may cause some temporary elevation of temperature. Diarrhœa and vomiting may be absent altogether, or there may be some slight increase in the frequency of the stools and some alteration in their character owing to the passage of an increased quantity of mucus. Some of the infants who pass into this state are found to be suffering from otorrhœa, and there is much reason to believe that this, in many cases, depends upon tuberculous disease of the middle ear. Tuberculous meningitis may ensue upon this otitis, or may come on without any such antecedent. Frequently, however, no complication develops, but the patient, in spite of the most careful feeding and nursing, grows gradually thinner and weaker, and is presently found dead in its cradle or even in its nurse's arms. In other cases death is determined rapidly by tuberculous meningitis, or broncho-pneumonia, or adenitis of the tracheo-bronchial glands. After death tuberculous disease may be found very widely distributed in the lungs, glands, spleen, liver, meninges, and more rarely in the kidneys, intestines, and thymus gland.

The diagnosis must generally rest largely upon the exclusion of other causes of progressive emaciation, such as insufficient food, chronic enteritis, relapsing broncho-pneumonia, and the condition of feebleness and imperfect powers of assimilation observed sometimes in children born before term. The cachexia of congenital syphilis may occasionally cause some hesitation, but the emaciation is not so extreme, polyadenitis (axillæ and groins) is not present, and the skin, even if it presents no characteristic or suspicious lesions, has a subicteric tinge. The existence of chronic ear disease will tend to confirm the diagnosis of tuberculosis.

The prognosis is unfavorable, although cases in which the diagnosis has been made occasionally recover, at least temporarily. If the hygienic surroundings of the patient can be made satisfactory, and if treatment leads to a gain in weight, some hope may be entertained of eventual recovery.

Treatment must be directed to maintaining nutrition, by careful dieting, by keeping the child out of doors for many hours a day, by free ventilation of the nursery, and by the administration of cod-liver oil, malt extract, or syrup of the phosphate of iron. The cases which recover, however, are usually those which are able to digest cod-liver oil.

Tuberculosis of **bones and joints** is the commonest localization of the disease in early childhood and has been estimated (Brandenburg)

to constitute 43 per cent. of all examples of tuberculous disease met with under four years of age. The consideration of this division of the subject falls to the surgeon; but it may be here remarked that surgical interference is in some cases, happily comparatively rare, followed by the outbreak of general tuberculosis or tuberculous meningitis.

Lymphatic Glands.—Chronic tuberculous disease of the **cervical glands** is one of the commonest forms of struma, as is indeed shown by the term "The King's Evil" applied to it when the royal touch was believed to be a sovereign cure for scrofula. In the majority of cases the specific infection is, no doubt, secondary to a simple adenitis, due to disease of the naso-pharynx or tonsils, or of the scalp (impetigo, eczema, pediculi), or of the teeth, but in others it is primary. Whether preceded by simple adenitis or not, the tuberculous infection is in the majority of cases derived from the tonsils and naso-pharynx. Tuberculous lesions of these parts are not a necessary antecedent, though chronic tonsillitis and adenoid vegetations, if not, as Dieulafoy supposes,[1] actually due to tuberculous infection, undoubtedly favor infection in two ways: (1) By the retention of the tubercle bacilli in the follicles of the tonsils or among the vegetations; and (2) by diminishing in these parts the activity of the phagocytosis upon which the destruction of bacilli depends. The enlargement of the glands is indolent, and retrogression and recovery the rule; but an attack of an acute infectious disease, such as measles, or of acute tonsillitis, or a local injury, or any cause producing deterioration of the general health may determine suppuration. A further risk is the extension of the tuberculous disease to the tracheo-bronchial glands, and thence to the lungs.[2] The enlargement of the glands may be enormous, obliterating the normal outlines of the neck and producing that resemblance to the thick neck of the pig which is said to be the origin of the term scrofula.[3] The glands primarily involved in carious teeth are those immediately beneath the jaw. Frequently the axillary glands are involved along with the cervical, and a continuous chain exists under the clavicle and the pectoral muscle.

The medical *treatment* of this condition resolves itself practically

[1] *Bull. de l' Acad. de Med.*, 1895, Av. 30.

[2] Eustace Smith (*Lancet*, 1895, vol. i., p. 1299) has made an ingenious suggestion as to another mechanism which may favor tuberculous infection of the lungs in cases of adenoid vegetation. He argues thus:—"Inspiration is inhibited by stimuli passing upwards through the superior laryngeal and the glosso-pharyngeal nerves; irritation continually applied to the periphery of these nerves must greatly restrict the admission of air to the lungs; the parts of the lungs least expanded will be those beneath the most flexible parts of the chest wall—the infra-mammary and supra-clavicular spaces: since the lungs cannot work fully, they cannot develop fully, and 'small lungs are always vulnerable lungs,' therefore the liability to pulmonary phthisis is increased."

[3] Lat. *scrofula*, a little pig, diminutive of *scrofa*, a breeding sow, literally a digger, from the habit of swine. (Skeat.)

into the treatment of those local conditions which produce adenitis of the cervical glands. The fact that lesions of the naso-pharynx, of the scalp, and of the teeth are capable of producing a chronic adenitis which is very liable to become tuberculous, lends a special imporance to the early and persevering treatment of such affections.

The tracheo-bronchial glands are the seat of tuberculous disease very frequently, and this condition is of great importance in relation to both general and pulmonary tuberculosis.

The trachea near its bifurcation and the large bronchi are in intimate relation with a large number of lymphatic glands. They may be divided into three groups: (1) The tracheal, on either side of the windpipe. (2) The tracheo-bronchial, which lie in the angle of bifurcation of the trachea and along the main bronchi; their most important relations are, in addition to the bronchi above, with the pulmonary veins below, and with the œsophagus, aorta, and posterior border of the lung behind. (3) The peri-bronchial, which are in contact with the main bronchi, their first subdivisions at the hilum of the lung, and with the bronchi as far as the fourth subdivision. These groups form one system massed about the end of the trachea, which corresponds with the third dorsal vertebra behind, and the junction of the manubrium with the gladiolus in front. The deep lymphatics of the neck, which lie both in front of and behind the carotid sheath, are continuous with those about the bifurcation of the trachea, and both with the subclavicular glands. In four out of five cases of tuberculosis in children the bronchial glands are affected, and in many cases it appears almost certain that this adenitis was the primary lesion (Fig. 12). These glands receive all the pulmonary lymphatics, and no doubt, in many cases, they are infected by tubercle bacilli, which have reached the lungs with the inspired air and have been carried back to the lymphatic glands. It has already been pointed out, in the remarks on etiology, that the bronchial glands may also become infected by gradual extension, in continuity either downwards from the cervical glands or upwards from the mesenteric, the infection being derived from food.

The affected glands become enlarged. In many cases, no doubt, especially after whooping-cough or measles, the enlargement is due, in the first place, to a simple adenitis which precedes and paves the way for the occurrence of tuberculous infection. The swollen glands are firm, and on section show a surface more or less pigmented according to the age and place of residence of the patient, but on the whole are semitransparent with one or more caseous patches. The caseous areas may be large, or some may have broken down into cavities containing a puriform fluid. In other cases of old standing, there may be more or less calcification, which may even be so extensive that the whole gland is transformed into a shrunken and calcare-

ous mass. An enlarged gland may become adherent to surrounding structures, and if a cavity has formed in the gland it may empty its puriform contents into one of the adjacent hollow organs. Rupture into the pulmonary artery causes sudden, profuse, and fatal hæmorrhage; rupture into a bronchus leads to the formation of a cavity, the walls of which may consist in part of the altered lung substance, and in part of the remains of the gland; rupture into the pleura may lead eventually to pneumothorax; rupture may also occur into the peri-

FIG. 12.

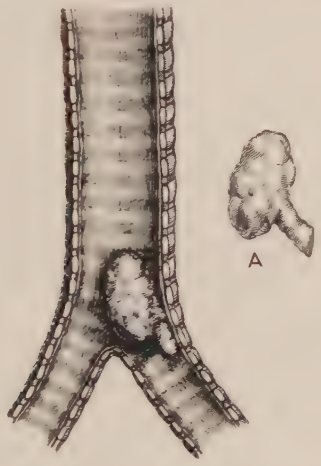
Section of the lung made through a mass at the root consisting of enlarged cheesy glands, blood-vessels, and bronchi. The section has passed also through cheesy masses within the lung. The infection in these situations is evidently of some standing. The cut surface of the lung shows, also, numerous recent tubercles, and similar tubercles are seen also on the dark surface of the congested pleura. One-quarter natural size. (After NORTHRUP.)

cardium, into the œsophagus, or into the trachea. The accident last named may cause sudden death, owing to impaction of a portion of the caseous gland in the trachea (Fig. 13). This has occurred in an infant of one year,[1] but most of the recorded cases have been from three to twelve years. A smaller mass carried into a bronchus may cause acute localized bronchiectasis.[2] It is probable that in some

[1] R. W. Parker, *Clin. Soc. Trans.*, Vol. xxiv., p. 6.
[2] Shaw, *Trans. Path. Soc.*, xxxviii., p. 90.

cases the entrance of caseous material from the glands into the trachea or bronchi has, by its aspiration into the bronchioles, determined the outbreak of a widespread and acute tuberculous broncho-pneumonia. Geill, in the series of cases already referred to, met with perforation of a bronchus by a softening gland four times. Northrup's [1] researches certainly support the view that in infancy and early childhood tuberculosis of the lungs is, in the great majority of cases, either a part of a more or less widely disseminated affection, or is secondary to tuberculosis of the bronchial glands. The tuberculosis extends to the lungs usually by continuity; the gland becomes adherent to the

FIG. 13.



The trachea and main bronchi from a case in which death was due to sudden dyspnœa produced by the rupture and dislocation of a caseous bronchial gland into the trachea. The spot at which the bronchial gland penetrated the trachea is shown, and at A the gland detached, *post mortem*, with its pedicle, consisting of thickened capsule by which it was tethered so that its removal was impossible, even after tracheotomy (Mr. R. W. Parker's case). Such a rupture is not very uncommon, though the mode of death is rare.

lung, and the tuberculous process overflows, as it were, from the glandular to the pulmonary tissue. The tendency to generalization from the glands directly appears to be small; the infection, as a rule, passes first to the lungs, and becomes generalized from thence by way of the blood (Fig. 12).

The physical signs of the enlargement of the tracheo-bronchial glands are diminished resonance in the interscapular region, especially on the right side, and blowing or harsh respiration in the same region. The symptoms are: (1) paroxysmal cough resembling that of whooping-cough, and ending often in vomiting, but without well-

[1] *New York Medical Jour.*, 1891, Vol. liii., p. 201.

marked crowing inspiration; (2) dyspnœa on exertion with slight cyanosis, and attacks resembling asthma. Simple adenitis of this group of lymphatic glands occurs most often as a sequel of measles or whooping-cough, and the symptoms, varying in severity from time to time, may persist for two or three months. Chronic adenitis of the tracheo-bronchial glands in children is, in most cases, tuberculous, and other forms of mediastinal tumor are exceedingly uncommon in early life.

The diagnosis is extremely uncertain; in many cases there are no symptoms, or they are ambiguous, and, with the physical signs, rather suggestive of broncho-pneumonia. Dyspnœa, produced by pressure on the trachea, may simulate bronchitis, with which it is often associated. The paroxysmal cough is not usually attended by inspiratory stridor and true whooping, but the resemblance of the symptoms to those of whooping-cough is so close that Guéneau de Mussy advanced the opinion that whooping-cough was due to a specific inflammation of the tracheo-bronchial glands. The history of contagion, and of the mode of onset of whooping-cough by a stage of fever antecedent to the whooping stage, as well as the characters of the cough, will, in a well-marked case, serve to distinguish the two conditions. In some cases, again, the paroxysms of dyspnœa suggest those of asthma, which is, however, a rare disease in early childhood. When there is hoarseness as well as dyspnœa the question of tracheotomy may arise, and since sudden dyspnœa in a young child may be due to a prolapse of a caseous gland, the operation ought to be performed, as it seems possible that under favorable circumstances the *débris* of the gland might be coughed up.

The prognosis of tracheo-bronchial adenitis, when the glands have become sufficiently enlarged to permit a diagnosis to be made, is grave, though there is no doubt that in less severe cases the tuberculous process frequently undergoes regression.

CHAPTER XII.

TUBERCULOSIS OF THE ABDOMINAL ORGANS.

Tuberculosis of the Mesenteric Glands—Tuberculosis of the Peritoneum: Acute, Chronic—Stomach—Spleen—Liver.

TUBERCULOUS, or "scrofulous," disease of the **mesenteric glands** was formerly supposed to occur very frequently in infancy and early childhood as an independent disease, and to be a common cause of marasmus. The popular term "consumptive bowels" includes tuberculous enteritis, peritonitis, and adenitis, but, in the majority of cases, the disease is a non-tuberculous chronic enteritis or entero-colitis. The mesenteric glands may become infected, either secondarily from tuberculous lesions of the intestines or peritoneum, or in the manner already described by the direct transmission of the virus contained in the food. The glands become enlarged in the course of typhoid fever, and in some cases of scarlet fever and measles. This adenitis quickly subsides, as a rule, but, as in the case of the tracheo-bronchial glands, it may pave the way for tuberculous infection, or light up a latent infection.

Tuberculosis of the mesenteric glands, unless and until the glands attain a large size, produces no characteristic symptoms; wasting, alternate constipation and diarrhœa, and tympanites can only reasonably be attributed to tuberculous disease of the mesenteric glands if the glands can be felt through the abdominal wall. This, even when the glands attain a large size, and form by agglomeration a tumor, which may be as big as a first, is not always possible, owing to the gaseous distension of the intestines.[1] The pressure of such a mass upon the vena cava may cause œdema of the lower extremities. It seems doubtful, however, whether so considerable an affection of these glands ever exists without tuberculous disease of the intestines or peritoneum, or both. The main importance of tuberculosis of the mesenteric glands is that the tubercle bacilli derived from the food find there a nidus in which they multiply, and from which they may be disseminated especially through the tracheo-bronchial glands to the lungs.

[1] Henoch, for example (*Vorlesungen ü. Kinderkrank.*, 6te Auf., 1892, s. 554), mentions a case in which a tumor of this nature, larger than a child's head, could not be perceived during life.

[Worcester has seen cases of tuberculosis of the mesenteric glands, which have been regarded and treated as "chlorosis," and the true nature of which he discovered only on subjecting the patient to the tuberculin test (vide infra). He earnestly advocates this test in all cases of suspected tuberculosis.]

Tuberculosis of the Peritoneum.—The peritoneum may become infected as part of a general tuberculosis, the infection reaching the serous membrane by the blood stream. More often it is carried by the lymphatics, either from the pleura, or from the genito-urinary organs, both rare events in children, or from the intestines. In the last case, which is the common mode of infection, there may or may not be tuberculous disease of the intestines.[1] In many, probably in the majority of cases, the intestinal mucous membrane shows no lesion, or evidence only of catarrh, which is often secondary. Repeated catarrhal attacks are, however, among the predisposing causes of tubercle of the peritoneum, since they weaken the resisting power of the intestinal epithelium. In these cases the tuberculosis may be —at first, at least—limited to the peritoneum.

Three types may be distinguished: (1) Miliary; (2) caseating; (3) fibrous.

Acute miliary tuberculosis of the peritoneum is generally a part of a general tuberculosis, or is associated with tuberculosis of the pleura. The surface of the peritoneum is studded with small gray tubercles, closely set. The serous membrane itself is usually inflamed, its surface has lost its polish, and soft adhesions have formed between adjacent organs and coils of intestine, but the cavity of the peritoneum is not obliterated; on the contrary, it contains usually a large quantity of clear yellow or greenish fluid. The mesenteric glands are slightly enlarged, but soft and semi-transparent. At a later stage the serous surface is covered by layers of fibrinous exudation, which can be detached easily, bringing the tubercles with them.

The symptoms of this form are very apt to be misinterpreted. On the one hand, if the miliary tuberculosis of the peritoneum be a part of a general tuberculosis, the symptoms present a very great resemblance to typhoid fever. Owing to the fact that there is usually much tympanites, the presence of fluid is masked, while abdominal tenderness (which is often neither general nor very marked), the fever, and depression present a close resemblance to the conditions produced by typhoid fever. On the other hand, when the tubercle is confined mainly or entirely to the peritoneum the condition is apt to be taken for acute peritonitis. The latter mistake is almost impossible to be avoided, since the eruption of tubercles on the serous

[1] The cases to which the terms Tabes Mesenterica and Tabes Mesaraica were formerly applied were, as a rule, instances of chronic tuberculosis of the peritoneum or mesenteric glands.

surface determines an acute inflammation of the membrane, and the symptoms—pain, tympanitic distension, and ascites—are, in fact, due, in large part, at least, to the peritonitis, and not to the tubercle by which it has been produced. The temperature rises steadily, and does not show the regular remissions of typhoid fever. The pulse is rapid and thready. Evidence may frequently be obtained of pleurisy with effusion. The patient is obviously very ill, lies in bed on the back, with legs drawn up. Food is refused, vomiting is frequent, and the vomited matters become, after a time, bilious.

The prognosis is, of course, bad, but on the whole less unfavorable than in simple peritonitis of equal intensity. The majority of patients succumb within two or three weeks or a month; but recovery may take place, the peritonitis subsiding and the tubercles eventually undergoing a fibrous change.

Chronic tuberculosis of the peritoneum is usually of the caseous type. Numerous tuberculous masses of varying size are present in the peritoneum and in the false membranes by which it is covered. Adhesions form in many directions, and irregular cavities, containing a puriform fluid, sometimes stained red or brown by recent or old hæmorrhage, are produced. The larger caseous masses tend in time to soften and in their immediate neighborhood may be found recent crops of small gray miliary tubercle. In fatal cases tubercle is found also in other organs, especially the pleura. Cicatricial changes are associated with the caseation in all the more chronic cases, but in some instances, especially those in which the tubercle is more deeply seated in the wall of the intestine, fibrosis is marked from, perhaps, the earliest stage. In such cases the serous membrane and its overlying false membranes form thick masses, layers, and bands, with caseating tubercles in their substance. The coils of intestine may be everywhere adherent, and separated only with difficulty. Perforation of the intestine may take place either into an adjacent coil or into a space among the coils, which is thus converted into an abscess cavity. In other cases the intestines, matted together by adhesions, but without any extensive adhesions to the parietal peritoneum, are, in the process of fibrous contraction, drawn back towards the vertebral column, forming a mass no larger than the fist. The mesenteric glands are enlarged and caseous. They may break down, and eventually discharge through the peritoneum; or they may, owing to fibrosis, become shrivelled, a process which is often accompanied by a cretaceous change.

The **symptoms** of chronic tuberculosis of the peritoneum present great variety. As a rule, the onset is very insidious, and advice is sought because the belly has been noticed to be growing large while the child has grown thin and anæmic, and has lost appetite. The abdomen is dome-shaped, with the umbilicus, which is often everted,

sometimes flattened, at the apex. Even in the early stage the contrast between the prominent distended abdomen and the pinched face and wasted limbs is often very characteristic. The abdomen is usually resonant throughout, fluid, if present, being masked by the gaseous distension of the intestines. Tenderness may or may not exist, but is seldom a prominent symptom. Attacks of colicky pain occur in many cases, and sometimes this is the first symptom noticed. During the course of the case the aspect of the abdomen may vary very much in relation with variations in the physical conditions. With a diminution in the amount of gaseous distension it may be possible to feel the irregular thickening of the omentum, and of the peritoneal surface of the intestines, and the enlarged mesenteric glands. These thickenings are, however, less easy to perceive than might be supposed. The liver may be enlarged, and the spleen is always affected, though it may not always be possible to detect any swelling during life. Great variations are to be met with in the quantity of fluid effused. In some cases the effusion is copious and is easy to be recognized ; in others it may be impossible throughout the case to be certain of its presence. In others, again, it may be detected at one time, its absorption may be observed, and later a fresh effusion may be discovered. In the absence of other well-marked signs of tuberculosis this disappearance and reappearance of peritoneal effusion is a valuable sign, and should raise a suspicion of tuberculosis. Extensive effusion, free in the cavity, may be caused by intercurrent peritonitis, or by a fresh eruption of miliary tubercle on the peritoneal surface, but is, in some cases, due to cirrhotic or interstitial tuberculous disease of the liver. Localized collections of fluid, so-called encysted peritonitis, are difficult to recognize, and commonly it is not possible to do more than surmise their existence. They are usually purulent, and are often associated with perforation of the intestine. In a minority of cases the fluid burrows towards the surface and eventually finds exit by the umbilicus ; or points, generally in the umbilical but occasionally in one or other iliac region. Tuberculous ulceration of the intestine is a common complication of tuberculous peritonitis, at least in its later stage, and the perforation may take place from within. Frequently this accident is unrecognized during life, the fæcal effusion and the resulting peritonitis being limited by the adhesions between the coils of intestine. Perforation may also occur from the peritoneum into the intestine, leading, perhaps, to the emptying of a localized collection of fluid into the gut. In other instances the ulceration into the intestine establishes a fistula between two coils. In either case the invasion of the intestinal mucous membrane causes severe diarrhœa, by which the patient's strength may be rapidly exhausted. In the later stages of a chronic case great recession of the belly may attend retraction of the intestines

towards the vertebral column. Through the wasted abdominal walls the agglutinated intestines may then be felt as an irregular tumor of doughy consistence, in which are embedded hard plaques or bands. It is not uncommon to find the upper part of the abdomen thus retracted while the hypogastrium is distended and contains fluid. The fever attending tuberculosis of the peritoneum is irregular; for weeks together no elevation of temperature may be noted, and then with some aggravation of the local symptoms, due perhaps to a fresh outbreak of tubercles or to localized suppuration, pyrexia may set in and continue for a long period. With caseation and suppuration the fever tends towards the hectic type. The patient grows weaker, and more emaciated; the skin assumes an earthy tint; the extreme emaciation of the lower limbs may be masked by œdema, and the skin is rough and dry except when, with a sudden fall of temperature after a hectic rise, it is for a short time drenched with sweat. The pulse is soft, small and frequent; appetite is lost completely. Vomiting, determined by the ingestion of even small quantities of liquid food, is frequent, and profuse diarrhœa often ensues to still further undermine the strength. The patient is bedridden; and sores on the sacrum, hips, shoulders, and elbows can hardly be prevented by the most careful nursing.

Prognosis.—The general tendency of chronic tuberculous peritonitis is towards death by exhaustion, owing to the interference with nutrition, or by involvement of other organs, especially the lungs. The patient is also liable to internal strangulation by bands, or to obstruction by compression or kinking of the gut. In some cases, which present all the early symptoms of tuberculosis, the abdominal distension decreases, and finally disappears, the nutrition improves, and the child makes, apparently, a complete recovery. When the later stage of general adhesion of the intestine, with retraction, and the formation of local purulent collections, has been reached, the prognosis is very bad, though evacuation of the pus either spontaneously or by operation, is followed occasionally by recovery if continuance of the suppuration can be avoided. The chance of recovery depends to a large extent upon whether the intestinal mucous membrane has escaped; if it has, there is some hope, if suppurative fever be absent also, that nutrition may be maintained.

Medical Treatment of peritoneal tuberculosis, whether acute or chronic, cannot be more than palliative, and must follow the same general lines as in simple acute or chronic peritonitis (*q. v.*). The question of the desirability of performing laparotomy, whether preceded by paracentesis or not, is the more pressing since there is evidence that the withdrawal of fluid effused into the cavity of a serous membrane affected by tuberculosis favors the arrest and retrogression of the tuberculous process. The number of cases mostly, however,

in women, in which this operation has been performed for tuberculous peritonitis is now considerable, and apparent recovery has ensued in about a quarter. The results in children have not been so good, but the number of reported cases under ten years is very small. The best results are likely to be obtained in cases in which there is a good deal of ascites, and if the operation is undertaken early, before adhesions have formed. Puncture is often followed by local adhesive peritonitis, and if the tuberculous nature of the peritonitis can be established it seems probable that laparotomy would give better results if performed at once, or, at most, after a single paracentesis. Henoch, who recommends frequent puncture and is not disposed, as a rule, rashly to resort to surgical methods, yet expresses the opinion that, in all cases in which, after treatment for four weeks and several punctures, no improvement has taken place, an exploratory laparotomy should be performed. When there is evidence of localized suppuration there will be less hesitation in adopting surgical treatment. In chronic tuberculosis of the peritoneum every effort should be made to maintain nutrition. With this object in view the patient should have the enjoyment of pure air and sunlight for as many hours of the day as possible; while the avoidance of exertion is desirable, confinement to bed or to the house has a most injurious effect on the general health. The abdomen should be covered with a flannel bandage, and, when warmly clad, the patient can be out of doors in a reclining carriage or on a couch for the greater part of fine days even during an English winter. The advantages of a warmer winter climate are, however, evident. The diet should be as ample as can be digested, and of the kind recommended in intestinal tuberculosis, with which peritoneal tuberculosis is, sooner or later, so commonly complicated.

Tuberculosis of the intestines is less common in children than in adults. It may be primary, the infection being derived from the food, or secondary to, as a rule, pulmonary tuberculosis, the bacilli being carried to the intestines by the sputum which is swallowed. When tuberculosis becomes generalized, the intestines may be involved along with other organs.

The conditions which exist in the intestines are not favorable to the growth of the tubercle bacillus. Prolonged contact with the mucous membrane, or some injury to its epithelium appear to be conditions necessary to the occurrence of infection. The first condition is fulfilled in the lower part of the ileum, immediately above the ileo-cæcal valve, and tuberculous ulceration is most often found in this situation, the second, by antecedent enteritis. The infection may become established first in the lymphatics which accompany the blood-vessels in the walls of the intestine, in Peyer's patches, or in the solitary glands. The effect is to produce granulomatous

thickenings which are in the first case annular, in the second longitudinal, and in the third small, round, and scattered. The granulomatous tissue undergoes caseous degeneration, and breaks down, leaving ulcers which have, at first, one or other of the forms indicated. Extension may take place by the formation of fresh granulations about the ulcers. In children the parts most often affected are Peyer's patches, and the typical annular ulcers are therefore less often seen in them. The ulcers have a thick irregular edge and a coarsely granular surface. Their depth varies in proportion to the amount of attendant thickening and tuberculous infiltration. The muscular coat may be involved, and eventually the continuity of the intestinal wall may be maintained only by the thickened peritoneum. Actual perforation is a rare event, especially in children. Cicatrization may occur and may entail extreme constriction of the gut. The large intestine is not often affected, but it is not uncommonly the site of a more or less acute and extensive catarrhal inflammation, with shallow ulceration, the specific nature of which is doubtful.

The **symptoms** of tuberculous ulceration of the intestine are far from characteristic, and its existence during life is more often suspected than proved. Whether the intestinal disease be primary or secondary, the first symptom to attract attention to its existence is usually diarrhœa. The motions at first are not very frequent, perhaps night and morning a soft, light-colored stool is passed. Gradually the motions become more numerous and fluid, and darker in color, until finally they are very dark brown, or even tarry, owing to bleeding from the ulcers. In its later stages the diarrhœa is very profuse, watery, and can be retained with difficulty. The odor of the stools is horribly offensive. The bacillus has been found in the stools. The abdomen is not constantly either distended or retracted. Tenderness may be elicited by firm pressure, or by gradually making deep pressure, and then removing the hand suddenly. Sometimes there is much colicky pain, and in some cases abdominal pain of a neuralgic character is an early symptom. It is not uncommon to be able to elicit a history of an attack of acute diarrhœa with colic some weeks or months before the onset of the persistent diarrhœa. This early diarrhœa has been attributed, with much probability, to the irritation produced by the first formation of tubercle; the final diarrhœa being due to the consequent ulceration. Emaciation and loss of strength are rapid and progressive unless the diarrhœa can be controlled by treatment. The fever produced by tuberculous ulceration of the intestines is of the hectic type; usually it is not high, and when the disease occurs as a complication of pulmonary tuberculosis it may not produce any recognizable effect on the temperature curve.

The **diagnosis** must depend to a great extent upon the recognition

of tuberculosis elsewhere. The existence of tuberculous enteritis may be assumed with tolerable certainty when diarrhœa, having the characters mentioned, sets in in the course of tuberculosis of the lungs or peritoneum. When, however, the disease of the intestines is primary, the diagnosis cannot be made with confidence.

The prophylaxis of tuberculosis of the intestines, owing to the serious and intractable nature of the malady, is of great importance; the liability of infection from the food, to which children fed mainly on milk are especially obnoxious, and the means by which it may be avoided, have already been mentioned. Infection by the sputum from tuberculous lungs should be guarded against by teaching the patient to expectorate what it coughs up into the pharynx, a difficult matter in young children. The propriety of the early and systematic treatment of intestinal catarrh will be obvious, since the liability to tuberculous infection is increased by the epithelial lesions which it produces. The child should wear a flannel bandage or cholera belt over the belly, and the thighs and legs should be warmly clad. The treatment of the disease when thoroughly established must be mainly palliative, and the remedies at our command are the same as those used in chronic enteritis.

Bismuth is the most valuable remedy. It may be combined in a mixture with tincture of opium or compound tincture of camphor, and should be given in frequent doses. When the pain is severe, morphine may be administered by hypodermic injection and hot fomentations or poultices applied to the belly; when the pain is accompanied by intractable diarrhœa small enemas of starch and opium (ʒj to ʒxj of starch freshly made, with tincture of opium ♏ v to xv, according to the age and general state) are useful to relieve both conditions. When tenesmus or the presence of much mucus in the stools points to affection of the large bowel, large injections of solution of nitrate of silver (1 per cent.) or sulphate of zinc have been recommended. The patient should lie on the left side, and be encouraged to retain the solution—which must be injected very slowly—as long as possible. Hayem has had good results from the administration of lactic acid by the mouth (♏ iij every three hours, increased gradually to thrice that quantity). Debove states that talc finely powdered, and given stirred up in milk, to the extent of an ounce or more during the day, sometimes has the effect of arresting the diarrhœa. If it subside, cod-liver oil in mixture, guarded by a small dose of opium, is often well borne. The diet should be simple and nourishing. Milk, sterilized unless it can be obtained from an irreproachable source, is the best. It should contain all the cream, or it may be diluted with an equal quantity of lime-water or whey, and the amount of cream made up, if it is found that the patient can digest it. Koumiss and Kephyr and aërated milk are valuable sub-

stitutes when milk is not well borne. Beyond milk the diet should consist mainly of meat in any form in which it can be best taken. Fats, such as butter and fat bacon, should be taken as freely as possible, but, they are not well borne when the temperature is elevated or diarrhœa severe. Vegetables, especially green vegetables and legumens, should be forbidden; they are not easily digested and tend to produce flatulence, which is a source of pain and danger. Potatoes, bread, and porridge, and other similar foods should be given with caution; and bread in small quantities, toasted, or, in its place, well-made friable biscuits. Sound fresh fruit, if freed from indigestible parts, can often be taken with advantage and without discomfort.

Tuberculosis of the Stomach has been observed in children, but is very rare. Tuberculous granulations form in the mucous membrane, caseate, and break down, forming round ulcers which bleed easily. The symptoms are pain, vomiting, and hæmatemesis, which may be very copious, and cause death rapidly. The intestines, and in some cases the peritoneum also, present tuberculous lesions.

It is convenient to add here that tuberculous ulcerations of the **tongue** and of the **palate** may occur in childhood, but they are extremely rare. On the tongue the ulcer is deep, with sharp edges, a yellow and sloughy base, and more or less surrounding induration. The prognosis is extremely bad, general or pulmonary infection being the sequel in most cases. The treatment which offers most hope of success is the excision of the ulcer, or scraping, followed by applications of chloride of zinc.

The **spleen** in acute tuberculosis is invariably the seat of tubercle, and in a very large proportion of cases of chronic tuberculous disease of the lungs, peritoneum, intestines, and meninges, it contains tubercles, which, however, may not be perceptible to the naked eye. Acute or extensive tuberculosis of the spleen is attended by enlargement of the organ, but it may be affected without the enlargement being sufficiently extensive to be recognized during life.

In acute miliary tuberculosis the **liver** seldom escapes in children. In peritoneal tuberculosis the infection may penetrate the liver from the surface, while in tuberculous ulceration of the intestine the infection may be carried to the liver by the portal blood. The tubercles may be disseminated irregularly through the liver and without relation to the portal, hepatic, or biliary vessels. This is the case generally in miliary tuberculosis, in which, moreover, the tubercles are commonly too small to be seen by the naked eye in the fresh organ. The tubercles may, however, be in relation with the portal system, and may then be accompanied by fatty degeneration. Tubercles of various sizes and in various stages of development may be met with also beneath the peritoneal covering, and in the portal

canals. Tuberculosis of the portal canals may determine an overgrowth of connective tissue and a form of hypertrophic cirrhosis, in which the liver is large, firm, and rather pale on section, and presents, in addition to the fibrous overgrowth which penetrates into the lobules, a fatty infiltration of the hepatic cells. The cell is distended by one or more large oily drops which have pushed the nucleus and protoplasm aside. These changes may be general or limited to certain areas, giving the liver a marbled appearance on section. Tuberculous fibrosis of the portal canals may also produce atrophic cirrhosis.

The **symptoms**, except in the rare cases in which atrophic cirrhosis develops and causes ascites, are not well marked. In hepatic tuberculosis, occurring in the course of acute general tuberculosis or of peritoneal tuberculosis, there is some enlargement of the organ, which may be tender; but, as a rule, symptoms referable to the liver are not well marked, though an icteric tint of the skin may develop. Hypertrophic tuberculous cirrhosis causes considerable enlargement of the liver, which is firm and has a distinct rounded edge and smooth surface. The spleen is enlarged. There is ascites. The urine is scanty and dark. It contains urobilin, often sugar, in proportion related to the carbohydrates of the food, and a diminished proportion of urea, while a trace of albumen may be present. Marked jaundice is rare, but the skin has an earthy tint, and there is often slight cyanosis. The face is puffy, and there may be œdema of the lower extremities, upon which petechiæ may form. Hæmorrhage may occur from the nose, stomach, or lower bowel, and the epistaxis in particular may be copious and obstinate. Sooner or later the lungs become infected, and many patients succumb to a rapid form of pulmonary tuberculosis. The temperature does not give any certain indications, and unless the lungs or other organs are affected there may be no fever, or only slight and temporary elevations of temperature.

The **prognosis** of tuberculous disease of the liver is exceedingly grave. As a rule, it is added to other tuberculous lesions, in themselves sufficiently serious. Hypertrophic fibrosis due to tuberculosis can only be recognized with certainty by its association with pulmonary tuberculosis, or by the appearances after death; so that it appears unprofitable to speculate as to the chances of recovery from this affection. For reasons of the same order, the treatment of hepatic tuberculosis calls for little discussion. When part of a general tuberculosis it must be treated on general principles, and the treatment of hypertrophic fibrosis however determined, must be the same.

CHAPTER XIII.

PULMONARY TUBERCULOSIS.

Acute Pulmonary Tuberculosis—Acute Tuberculous Pneumonia—Acute Tuberculous Broncho-pneumonia—Chronic Pulmonary Phthisis—[Tuberculin]—General Remarks on Treatment.

Pulmonary Tuberculosis.—Pulmonary tuberculosis in children, as in adults, may be acute or chronic. It is relatively common in infancy, rare under four or five years, but frequent after that age.

Three types of *acute tuberculosis of the lungs* may be distinguished, presenting distinct pathological and clinical features: (1) If the infection be derived from the blood, the changes begin in the tissue of the alveoli, the capillaries, and the alveolar epithelium. The tubercles may be disseminated through both lungs when the pulmonary tuberculosis is often only a part of a general tuberculosis; or they may at first be confined to one lung, being either scattered more or less uniformly through it, or closely set in the upper or lower lobe. After the first outbreak the morbid process may be arrested, either temporarily or permanently, the individual tubercles eventually undergoing a fibroid change. More commonly the tubercles become caseous at the centre, while the tuberculous process extends at the periphery until by coalescence large tracts of the lung are involved, and undergo caseation and softening. (2) More rarely, the infection of one lung or one lobe is so intense that the pulmonary tissue becomes densely packed with tubercle. This is attended by catarrhal inflammation of the bronchioles and alveoli and œdema of the tissue intervening between the tubercles, so that an acute tuberculous pneumonia is produced. (3) If the infection takes place by way of the bronchi—"inhalation tuberculosis"—the resulting disease has the general pathological character of broncho-pneumonia. The tuberculous process is then primarily peribronchial, and the anatomical element involved is the lobule, though when the infection is intense so many adjacent lobules may be affected that the whole lobe, or a large part, may become consolidated. Retrogression and fibroid change may occur, but a far more common sequel of events is caseation, softening, and the clearing away of the disintegrated matter with the formation of vomica. This appears to be the form which pulmonary tuberculosis commonly

takes when it follows measles or whooping-cough, the bronchitis or broncho-pneumonia by which these specific diseases are so frequently complicated having prepared the bronchial and pulmonary tissues to receive the tuberculous infection. After measles, especially, it may be difficult to say at what stage the broncho-pneumonia becomes tuberculous. In some cases the child has already suffered from some chronic tuberculous affection of the lungs, the glands, or joints.

(1) **In acute pulmonary tuberculosis** there is a widespread irruption of miliary tubercles in the lungs, and the symptoms are those of the bronchitis thus produced. Cough is troublesome, and the expectoration, if it can be obtained, will be found to be muco-purulent, and perhaps tinged with blood. There will probably be no deficient resonance on percussion ; the note may be indeed tympanitic, a significant change suggesting the occurrence of emphysema to compensate for the areas of lung occupied by the tubercles, which, though individually minute, in the aggregate involve a large portion of the breathing space. Dyspnœa, which is a prominent, and often also an early symptom, must be attributed to the same cause. It is greater than can be accounted for by the physical signs, and these two conditions, if associated with some cyanosis of the lips, ears, hands, and feet, should excite suspicion of tuberculosis in any case in which the physical signs point to no more than bronchitis. The breath sounds may, however, be altered in some areas, generally at one base, or in the interscapular region, becoming harsh, bronchial, or tubular, and small crepitations, coarse mucous râles or sibili may be heard. Tubercle of the pleura may produce sounds which closely resemble true fine crepitations. The pulse is rapid, the temperature is 101° to 103° F., and the child is evidently far more ill than is to be accounted for by the extent of the physical signs. Enlargement of the spleen may be detected early in the most acute cases, and is usually to be observed at a later stage in the less acute.

The *prognosis* in cases of this type is extremely bad. Many ultimately present cerebral symptoms due to the infection reaching the meninges ; others succumb to the severity of the pulmonary disease within a fortnight ; others survive for a month or even several months. This is perhaps observed with special frequency in cases following typhoid fever.

(2) **Acute tuberculous pneumonia**, a rare affection at any age, is not often observed in children.

The **symptoms** are identical with those of acute croupous pneumonia, but crisis does not occur, the fever is usually less high, and after four or five days becomes irregular. Profuse sweats often occur at night, and there may or may not be diarrhœa. The persistence of the symptoms and of the signs of consolidation raise a suspicion of tuberculosis, and after two or three weeks, if the patient

lives so long, the signs of softening and of the formation of cavities may be discoverable. Before this, if the sputum is brought up, it will be seen that it is purulent and greenish, and the tubercle bacilli may be discovered, perhaps, in large numbers. The *diagnosis* from pneumonia in the early stage is impossible. Later the persistence of the signs and symptoms, the irregular fever, and rapid emaciation and loss of strength will justify an unfavorable diagnosis, even if tubercle bacilli be not discovered in the sputum, nor tubercle in the choroid. The occurrence of cerebral complications (meningitis) will often confirm suspicions already entertained.

(3) **Acute tuberculous broncho-pneumonia** is a common affection in children. It is the form which ensues most often after measles and whooping-cough. It is preceded in other cases by various exhausting diseases—gastro-intestinal disorders, chronic bronchitis, or repeated attacks of broncho-pneumonia; but it may come on suddenly in the midst of apparent good health. It is lobular in distribution, though by confluence large areas of consolidation may be formed. These undergo caseation and softening. The small bronchi, thickened by the tuberculous process, are filled with caseous material, but with the progress of the softening and the onset of suppuration they are destroyed; eventually small vomica or narrow anfractuous cavities are formed. These changes may be irregularly distributed through both lungs, or may be at first confined to one lung or to one part of it, generally the apex. In such cases death is often determined by the rapid infection of the other lung, so that while in one lung we find caseous areas and cavities, in the other we find numerous tubercles in various stages, the majority gray or miliary.

The signs and symptoms are those of broncho-pneumonia. The onset may be acute or subacute. In the former case the child, when convalescent from some febrile disease, while suffering from whooping-cough, or after perhaps a few days of peevishness and anorexia, becomes suddenly feverish (102°–104° F.) and suffers from troublesome cough. The respiration is hurried and the pulse quickened, but not in proportion, so that the pulse-respiration ratio is disturbed. The face is suffused or slightly cyanosed, and the skin is dry and harsh. The physical signs may be at first those of bronchitis only, with perhaps here and there, at the apices, at the posterior bases, or in the axillary regions, areas over which crepitant and small mucous râles are heard. The percussion note is usually unaltered, or in the regions where the finer râles are heard, a little high-pitched, with an increased sense of resistance to the pleximeter finger. If the apices are affected by scattered patches of broncho-pneumonic consolidation, they may be tympanitic. Later there may be distinct evidence of consolidation over more or less extensive areas, including generally the apices. The condition of the child deteriorates rapidly, and

emaciation may become extreme; there are sometimes copious sweats, and often diarrhœa. In cases of this class the patient seldom survives long enough for cavities to form. Death may indeed ensue in a few days, and the tuberculous nature of the disease may only be established by careful microscopical examination of the broncho-pneumonic areas.

Chronic pulmonary phthisis.—Chronic pulmonary tuberculosis (pulmonary phthisis), seldom seen in infancy, becomes after the age of three years progressively more common, until about the age of puberty it is not infrequent. It resembles in general characters the pulmonary phthisis of adults, and will not, therefore, be discussed at length here. The main peculiarities of chronic pulmonary phthisis in childhood are the frequency with which it is secondary to tuberculosis of the bronchial glands, and the fact that—perhaps in consequence of this—the earliest lesion in the lung is commonly not at or a little below the apex, but in the neighborhood of the hilum.

The symptoms are similar to those of the same disease in the adult. The chronic or insidious mode of onset is less common, for pulmonary phthisis in children ensues most often upon an attack of severe bronchitis, broncho-pneumonia, or pleuro-pneumonia. In such cases it is commonly impossible to determine whether or not the initial disease of the lung itself partakes of a tuberculous nature. When chronic pulmonary tuberculosis follows measles, the development of destructive lesions in the lungs may be very insidious, and the same character may belong to the disease when it develops in a child who has suffered from repeated attacks of bronchitis or gastro-enteritis. Hæmoptysis is very rare as an early symptom, and is not common in the later stages. Cough is often not severe, and there may be no expectoration, the sputum being swallowed. The child, after the acute illness—which, as has been said, usually marks the commencement of the disease—is found not to regain its former health. Though restless it is indisposed to play, is a little short of breath, and has a short, dry cough. It is anæmic, and there may be some general puffiness of the upper part of the trunk and of the face, which is pale. In the afternoon or evening there is some slight elevation of temperature, accompanied by a malar flush and increased restlessness and volubility, and followed by perspiration and chilliness in the early morning. With these symptoms there are usually some loss of appetite and slight emaciation. The physical signs, except in those cases in which the tuberculous disease runs on directly from antecedent broncho-pneumonia, are at first often very indefinite. A difference in expansion between the two sides is not usually to be recognized except in cases in which the phthisis succeeds pleurisy. Gentle pressure with the finger tips in the intercostal spaces may elicit some tenderness. The percussion note may be high-pitched in some areas,

especially, perhaps, in the interscapular area, in the axilla, or below the angle of the scapula. On the other hand, it may be, and often is, tympanitic at the apex in front, or, indeed, over the whole of one side, or over both backs. Vocal fremitus and resonance may be increased in those parts in which the resonance on percussion is diminished, or over wider areas, and the breath sounds may here be harsh and bronchial. A dry crackle may be heard at the end of inspiration, but will often disappear temporarily after a deep breath, or after crying or coughing. Later, moist râles may be heard, and may be observed with the lapse of time, to become larger and more clicking. Finally, cavernous respiration and râles may develop. Too much importance, however, must not be attributed to auscultatory signs of a cavity, since they may be simulated by bronchiectasis, which is produced rapidly in children. At the apex moist sounds, due to resolving pneumonia, sometimes, in thin children, have a hollow character, while at the same time the breath sounds are high-pitched so that a general resemblance to the physical signs of a cavity is produced. The dyspeptic troubles common in adults are often absent; on the other hand, it is not uncommon to find that the child is very subject to attacks of gastro-enteritis, or suffers from chronic entero-colitis, with frequent offensive mucous stools. If the pulmonary disease is very chronic, the general nutrition may suffer little, but such children are very liable to attacks of local pneumonia around the tuberculous area. These attacks are accompanied by high fever, dyspnœa, and a sudden extension of the physical signs. As the fever subsides, the signs diminish again rapidly, and the condition may seem to revert to that which existed before the acute attack. In children under three years of age, the temperature may be little, if at all, raised for long periods together; indeed a case may be under observation for weeks without any elevation of temperature being noticed. In other cases, and in older children as a rule, fever of hectic type is established early, though the maximum temperature may occur in the morning; altogether, the temperature curve in a case of pulmonary phthisis in a child, especially a young child, tends to be more irregular than in adults. As a rule, when the disease has reached a stage in which the physical signs enable a positive diagnosis to be made, the child rapidly becomes extremely emaciated. In some cases the appetite is retained to a wonderful extent into quite a late stage, and, in the absence of late diarrhœa, which is relatively uncommon in children suffering from phthisis, the nutrition may be maintained to a degree which may lead to errors in prognosis.

[**Tuberculin as a Diagnostic Agent.**—While the therapeutic value of tuberculin is still *sub judice*, its value in diagnosis must be admitted. This depends upon the fact that tuberculin when introduced into a tuberculous individual produces in a few hours a "reaction" char-

acterized by rise of temperature, chilliness, cough, and general malaise. No such reaction is observed in non-tuberculous individuals similarly treated. This test is now very generally applied in this country to all large herds of cattle, and its value to the public is universally acknowledged. Unfortunately the test is but little used with human beings on account of the prevalent fear of "unfettering" the bacilli from some latent focus with their subsequent dissemination over the whole system. Such dissemination, however, does not take place in cattle, nor did Koch, testing 1,000 cases in man, observe any such result. In view of these facts, a more extended use of this valuable preparation in cases of suspected tuberculosis is earnestly recommended, especially among children where the diagnosis is so often difficult or impossible in the earliest stages, the time at which treatment is most successful.

Too great care in the selection of a preparation cannot be exercised. It should be of known quality and strength. The amount to be injected is variously given by different observers as one-twentieth of one milligramme in infants, up to from one-half to ten milligrammes in adults. It may be injected in any part of the body, but must be injected deeply. A rise of temperature from 102.5° to 104° with other general symptoms within twenty-four to forty-eight hours should be considered as a definite test. If there be no reaction after the first injection, the test may be repeated with gradually increasing amounts at intervals of four or five days, till three or four trials have been made.]

General Remarks on Treatment.—The treatment of chronic tuberculosis, whether affecting the pulmonary or abdominal organs, is, in practice, most difficult. No drug has any power of arresting the progress of the infection; but since the destruction of tissue is produced not only by the specific bacillus but also by pyogenic organisms, antiseptic drugs have, under favorable circumstances, a certain influence over the disease as a whole. Of these the most valuable is creasote [which may be given as follows:

Creasoti,	♏ss
Spir. Rect.,	
Spir. Chloroform.,	āā ♏ijss
Tn. Card. Co.,	
Extract. Glycyrrh. Liq.,	āā ♏v
Mucil. Tragacanth.,	♏x
Aq.,	ad ℥j

Dose at 1 year. Appendix.]

On the whole, however, drugs are of value chiefly for holding in check secondary conditions which are undermining the strength. Thus morphine in linctus may be called for by cough which prevents sleep, bismuth by diarrhœa, belladonna or oxide of zinc by copious sweating.

[Liq. Morph. Hydrochlor., ♏i–iij
Acid. Hydrochlor. Dil., ♏j
Tn. Aurantii Rec., ♏xx
Glyc., ♏x
Aq., ad ℥j
For children over 8 years. Appendix.]

Arsenic is praised by Jacobi for its stimulating effect on cell-growth. He finds its "principal indication in the peculiar fragility of the blood-vessel walls resulting in pulmonary hæmorrhage." He gives two minims of liquor arsenicalis daily in three doses, largely diluted, after meals, to a child of a few years old. The drug may be continued for an indefinite period, unless symptoms of an overdose—gastric and intestinal irritation and local œdema—appear, as seldom occurs if small doses of opiates are given with it. With arsenic he combines also digitalis,[1] which he recommends (1) on account of its influence in favoring the excretion of the kidney and the emptying of the veins, thereby accelerating the flow of lymph and improving general nutrition; (2) because the pulmonary artery is relatively larger in childhood, so that any insufficiency in the heart muscle tends to produce œdema of the pulmonary tissue, a condition obviated by the improvement in the pulmonary circulation caused by digitalis; and (3) on account of its action as a cardiac stimulant, since this is attended by improvement in the nutrition and development of the heart, which is relatively small in phthisis. He continues the remedy for weeks or months, but, if a speedy action is required, combines with it strophanthus, sparteine, or caffeine.

The *diet* is an important element in the treatment of all forms of chronic tuberculosis. In pulmonary phthisis the appetite is usually small and capricious, while hectic fever and sweating, even if diarrhœa be absent, combine to reduce the patient's strength and to waste his tissues. In young children milk should be the staple article of diet, but care must be taken to obtain it from a source free from tuberculous taint. Meat, either raw or very lightly cooked and scraped to a fine pulp, is a valuable addition, and Debove praises a method of "forced feeding" by finely powdered dry meat introduced directly into the stomach. In older children a greater variety in the diet is requisite, and means must be taken to overcome the distaste to fatty foods, which is often very marked. Cod-liver oil is the most effective form in which fat can be given. It is best taken alone, or with malt extract, and if of good quality the patient, as a rule, soon becomes reconciled to its taste; indeed, many children acquire a great liking for it. When the repugnance to it is great it

[1] "Therapeutics of Infancy and Childhood," by A. Jacobi, M.D., etc., Philadelphia, 1896. He recommends, in preference to the infusion or tincture, which are often not well borne by the stomach, a liquid extract, or extract—♏ij of the former, or gr. of the latter, for a child a few years old, daily.

may be given as an emulsion, and then, after time, the pure oil will generally be tolerated. Eructations some little time after swallowing the oil, of which many patients complain more than of the taste of the oil itself, are best corrected by taking some carminative, such as peppermint, shortly after the dose of oil. Cod-liver oil should be given after meals, and must usually be intermitted during any febrile attack.

The point of prime importance, however, in the treatment of chronic pulmonary phthisis is that the patient should at all times breathe *fresh air;* other climatic conditions—temperature, moisture, elevation above the sea—are of secondary consequence to this. The patient should be under strict discipline as to habits of life, time spent in the open air, food, clothing, etc., and the necessary conditions are best fulfilled at a sanitarium, of which many exist in Germany. Prolonged treatment is necessary in most cases before any permanent improvement can be looked for. In the case of quite young children, or in older children when treatment in an institution is impossible, the patient should be put under the best obtainable hygienic conditions, should spend as much time as possible in the open air without fatigue; and the rooms used, especially the bedroom, should be kept scrupulously free from dust, and should contain no heavy hangings nor much furniture. The sputum should be disinfected and destroyed, and soiled linen should be disinfected. This prophylactic treatment is to be recommended both in the interest of the patient and of other members of the family. The patient should not be nursed by a person suffering from active phthisis, and the fact that consumption is a disease which may easily be communicated under ordinary circumstances of domestic intercourse between children and parents should be impressed upon the latter.

CHAPTER XIV.

SYPHILIS.

Inherited Syphilis; Infection; Symptoms; Lesions of Skin and Mucous Membranes, of Viscera, of Bones; Contagiousness—Late Syphilis—Diagnosis of Inherited Syphilis—Prognosis—Acquired Syphilis—Treatment of Syphilis.

Syphilis in infancy may be inherited or acquired. In the large majority of cases it is inherited.

Inherited Syphilis.—Infection of the ovum with syphilis, which may take place at the time of conception, may result in the death of the fœtus before birth, and consequent abortion; in the birth, at or before term, of a dead child; in the birth of a living child suffering from cachexia, with or without certain characteristic lesions of the skin; or, in the birth of a well-nourished living child, which a few weeks later presents the skin lesions, with or without marked cachexia.

For three, or at most five years after his chancre, the father may infect the mother in the ordinary way, and both parents then suffer from obvious syphilis. The father may infect the fœtus as late as twenty years after his chancre, when for years he has presented no manifestations of syphilis, and the mother may have a series of syphilitic pregnancies, resulting in miscarriages or in syphilitic infants, without at any time herself presenting any syphilitic manifestations; but she does not contract syphilis from her own child (Colles' law). In the same couple the severity of the infection transmitted to the fœtus tends to decrease with succeeding pregnancies. Thus it is the rule for a woman to have at first several abortions, then perhaps a child born dead, then a living child which suffers from inherited syphilis. Children born later usually suffer less severely; but this "law of decrease" (Diday) is not without numerous exceptions. Sometimes the third or fourth child suffers more than the second, and it has been alleged that in some families children of one sex suffer more severely than those of the opposite. In twin pregnancies one child may be affected while the other apparently escapes. The apparent escape of the mother of syphilitic infants by a syphilitic father has been accounted for on the supposition that she undergoes a mitigated infection derived from the fœtus,

but, as Coutts[1] has pointed out, the theory that she absorbs from the fœtus a syphilitic antitoxin would account not only for her own apparent immunity but also for the gradual decrease of the severity of the disease in later pregnancies. A man under proper treatment may beget a healthy child, and later, having given up treatment, an infected fœtus. If the mother be infected but not the father, death of the fœtus is the most likely event; but if the child is born alive it will probably suffer from inherited syphilis. If both parents have suffered from manifest syphilis, the chance of abortion or still-birth is greater. Practically, however, the question whether a child brought for treatment has derived its infection from father or mother, or both, is not one of much importance in prognosis, which must rest mainly upon the condition of the infant itself, especially as to nutrition.

The main **symptoms** are marasmus and anæmia and certain lesions of the skin and mucous membranes. At birth the infant may be well nourished or already puny and emaciated, but as a rule there are no distinct manifestations. If these be present, death ensues almost without exception in a few days. In a typical case the infant is fairly well nourished at birth, but does not thrive well, becomes anæmic before the end of the first month, and during the course of the second month begins to suffer, first from snuffles, and then from an eruption. The appearance of the symptoms may be delayed until the third month, or even to the sixth month; only in exceptional cases to a date later than this. An infant may be born, either alive or dead, with more or less extensive **pemphigus**, or it may be born without the eruption, which appears during the first, more rarely in the second, week of life. It comes out first on the palms and soles, or other parts of the feet and hands; the face is less often affected, the trunk rarely. The bullæ are surrounded by a red zone, or seated on a dusky red, slightly elevated base. Usually they are flaccid, and contain pus and blood; but in the less severe cases the fluid may be clear and the bullæ tense. The nail-bed is often diseased, leading to blackening and destruction of the nail, or to a deformity of the nail, which is narrow at the base and spread out like a fan at the free end. The bullæ, when they rupture or dry, form dark yellowish-green scabs, often, from confluence, of large size, under which a shallow unhealthy ulceration tends to spread. The child is usually marasmic at birth, or very quickly becomes cachectic, and the prognosis is extremely bad. This eruption, when well marked, indicates a very severe form of the disease, but Crocker[2] states that he has seen one

[1] "Some Aspects of Infantile Syphilis," London, 1897. These Hunterian Lectures contain an excellent discussion of many of the moot points as to syphilis, both inherited and acquired in infants.

[2] "Diseases of the Skin," London, 1893, p. 544.

severe case, in which the eruption was present at birth, recover under immediate mercurial treatment. Usually the infants succumb quickly to the cachexia. Occasionally cases are met with in which a few shallow bullæ appear as late as the fourth or fifth week of life. Such cases are amenable to mercurial treatment, and the prognosis is much better.

Fig. 14.

Syphilitic marasmus in an infant, showing the senile cast of countenance, rhagades about the mouth, emaciation, inelastic skin, scanty hair, and anæmic œdema of hands and feet. (From a photograph.)

The **marasmus** produced by inherited syphilis (Fig. 14) may be the first symptom in time as it is in importance. We may distinguish two factors—anæmia and wasting. The infant may be born marasmic, or it is born well nourished, but before the end of the first month begins to be anæmic. The skin has a faint yellow or straw-colored tinge, and looks semi-transparent, as though it were coated with a thin layer of yellow wax. When the affection is more profound the color is deeper, and the skin has an opaque brownish tint,

which has been compared to that of *café au lait*. The hair grows thin, and a slight branny desquamation, often accompanied by yellow stains may be seen about the scanty eyebrows. Usually the first local symptoms is snuffles, due to a lesion of the nasal mucous membrane, attended by much secretion and, after a time, by swelling, so that the nasal passages become blocked, and the infant, in consequence, has difficulty in suckling. Next, one of the various forms of skin eruption appears generally first about the buttocks or round the mouth. The anæmia, which is due to a decrease in the number of the red blood-corpuscles and in their hæmoglobin, is, in my experience, always present to some extent before the eruption appears. It is usually accompanied by wasting. If the infant is suckled at the breast, this may not be great; but in those fed artificially it is always considerable and often extreme. Marasmus may continue after the eruption and even the snuffling, which is more persistent, have disappeared under the influence of mercury. It may be the only symptom at the time advice is sought in cases in which the family history, or the history of snuffles and rash which passed away after a short time, leaves little doubt that the infant is the subject of hereditary syphilis. Again, obstinate anæmia, with or without wasting may be the only symptoms in the later born infants of a family known to be syphilitic.

If, either soon after birth or at a later date, the marasmus have become well established, it is too often the case that mercury has little effect. The intestinal mucous membrane is extremely wasted, so that the wall of the gut is as thin as writing paper, and to the naked eye seems to consist only of the peritoneal coat, with a very thin mucous lining. The liver also is often fatty, and it is clear that digestion, absorption, and assimilation are all most imperfect.

The lesion of the *skin* most often seen in the early stage is erythema, but papulo-squamous, papular, vesicular, or pustular eruptions may take its place. Later lesions of the mouth are mucous tubercles and the so-called syphilitic furuncle. The syphilitic roseola, which is the commonest eruption in acquired, is rare in inherited syphilis.

The conditions to which the term **erythema** is applied is a superficial hyperæmia with slight infiltration. It is commonly either limited to parts liable to irritation by discharges or clothing, or is most marked in these situations. The skin is reddened, a little thickened, and the finer natural markings are obliterated. It begins, as a rule, as patches on the buttocks and soles of the feet. It spreads by the formation of new patches in the healthy skin, which enlarge until they coalesce with each other and with the older areas. It extends often over the whole of the posterior aspect of the lower limbs and feet, so that the infant looks as if it had walked and sat down in a

puddle of red dye. It may extend on to the trunk, in a continuous sheet behind, but in patches in front, and its upper limit often corresponds distinctly with the margin of the napkin. On other parts of the trunk, at the folds of the axilla and of the neck, about the mouth and behind the ears, patches are often to be seen (Fig. 10). Occasionally the eruption extends over the whole body; infants thus extensively affected seldom recover. On parts of the skin moistened by perspiration or urine the surface is smooth, glistening, and of a red brown or coppery red color. On the drier parts the color is less deep, and there is some desquamation, which is usually detached in small flakes. On the soles of the feet it may separate in larger flakes, leaving the whole surface smooth, and deep red with a frill of half detached epidermis at the edge. An eruption which rather resembles this, but is held to correspond with the **papulo-squamous** eruption of acquired syphilis, consists of smaller patches, of a lighter red or yellowish color, with more infiltration and a more copious desquamation. Such patches tend to heal in the centre, and then present a thickened red edge, and a flat faintly yellow centre. When small, few in number, and situated on the face, temples, or neck, this condition has, it is said, been mistaken for ringworm. The patches may spread far and wide, coalescing at their edges, while healing at the centres, thus forming geographical patterns.

The eruption may be **papular**. The most characteristic form is a flat papule, roundish or of irregular angular outline, with a dusky red hue and a shining surface. The papules occur in groups or singly, generally on the neck, shoulders, or arms, and are often associated with erythematous or pustular lesions about the buttocks. In other cases, and, according to Crocker, more often, the papules are small, convex or acuminate, of a bright or brownish red, and crowned with a scale or a small pustule. They occur in irregular groups on the limbs, and are almost always in my experience associated with pustular or ulcerated lesions of the buttocks and face; and their specific nature is often doubtful—that is to say, they commonly appear to be due to secondary pyococcal infection in a syphilitic infant.

Upon parts which are constantly moist, and especially in infants who are not very carefully nursed, any of the skin lesions already mentioned may be complicated by suppuration. Thus shallow ulceration on the buttocks often occurs in erythema, and about the mouth crusts may form under which a creeping ulceration spreads slowly. Ecthymatous sores may form on the buttocks, face, or arm, and a greenish crust covers a sharp-edged ulcer, which exudes a thin greenish or sanious pus. These suppurating lesions are associated with secondary infection by pyogenic organisms, and the infective material carried to other parts of the body may excite a widespread impetigo.

The syphilitic eruptions when they fade leave some discoloration, the depth and permanency of which is in relation to the severity of the lesion.

The lesion of the skin to which Barlow has applied the term **syphilitic furuncle** is a manifestation which appears later than the eruptions hitherto mentioned, as late even as the fifth or sixth year. It is often met with, and is exceedingly characteristic. The term "blind boil" applied to it commonly by mothers well indicates its general character. An indolent swelling involving the whole skin forms slowly without any lesion of the surface, which, however, gradually assumes a purple color. The swelling, which eventually may be half an inch in diameter, then contains a few drops of thin pus. Untreated it may persist for months, finally undergoing retrogression, leaving some thickening and puckering of the skin. If irritated, it may break down at the surface, forming a shallow ulcer on a thickened base. Under mercurial treatment it disappears rather slowly, leaving no scar. The inner and outer aspects of the thighs and the front of the abdomen are the most frequent sites of these lesions. They are not pathognomonic, as similar cutaneous lesions may be observed occasionally in cases in which no history of syphilis can be obtained, but they afford very strong confirmatory evidence.

The *situation and distribution of the skin lesions* are determined to a very large extent by local sources of irritation (Fig. 15). It is for this reason that the buttocks, which many times a day are in contact with urine and fæces, and the lower lip, which is kept wet with saliva if the infant dribbles, are so frequently the parts first and most severely affected. If the parts are kept dry and clean, the skin lesions may even disappear without specific treatment; and, speaking generally, the skin lesions in infants who are well cared for are less extensive and persistent than in those constantly dirty.

The **mucous membrane** affected earliest is that of the nose. It becomes swollen, and a sero-purulent discharge is soon established, which excoriates the upper lip and cakes about the nostrils. The obstruction to respiration thus produced causes "*snuffles*," which commonly precede the eruption by a short time. If the nasal lesion be severe and long lasting, some arrest of growth of the cartilage ensues, so that the nose is stunted but broad at the base. Permanent deformity may result, but as a rule the nose gradually improves in shape as the child grows. Snuffles often persist long after the skin affections have disappeared under treatment. This is no doubt due, in part at least, to the great liability of the nasal mucous membrane in infants to catarrh, owing, perhaps, to the fact that they breathe altogether through the nose. Indeed, the acute coryza, which is very common in infants, and is often associated with laryn-

gitis, may, if the patient be seen first when the attack is subsiding, lead to an ungrounded suspicion of syphilis. The larynx is often affected soon after the nose, and the cry becomes toneless, hoarse, or, as it were, whispering. Sores in the mouth are rare in the early stage, but occasionally a superficial glossitis occurs simultaneously with the erythema of the buttocks, which indeed it much resembles; the tongue is of a uniform beefy-red color and smooth, or it presents a few very superficial linear ulcers. A little later, in association with either ulceration of skin lesions on the face or mucous tubercles

FIG. 15.

Drawing (semi-diagrammatic) of a well-nourished infant presenting an extensive, dry, desquamating syphilitic eruption, to illustrate the distribution of the eruption in regions specially liable to irritation by friction (upper arm, knees) or by secretions (buttocks, neck, mouth). The abrupt arrest at a level corresponding to the upper edge of the napkin is well seen.

in the mouth, shallow linear ulcerations affecting the red margin of the lips and the adjacent mucous membrane form, especially about the angles of the mouth.

Mucous tubercles are a later manifestation. They appear usually from the sixth to the twelfth month of life, and are apt to recur for four or five years. They are met with especially about the angle of the mouth and the anus. Associated with them deep cracks may form at the angle of the mouth, producing the well-known **rhagades**,

which are often very obstinate and leave permanent scars. At the anus the condylomata sometimes break down, forming very deep ulcers with thickened edges, which are concealed until the nates are separated; in other cases, in older children especially, cauliflower excrescences may form.

The **spleen** may be enlarged at the time of birth, and may so continue for many months; not very infrequently the enlargement commences later than the eruption, or after it has disappeared. Sooner or later it occurs in a large proportion of cases,[1] but after the first year, rickets is so common in children who have suffered from syphilis that the enlargement in them may with equal propriety be ascribed to the rickets. When the organ is enlarged the infant is always anæmic, but it may not be enlarged in extreme marasmus. The enlargement is due to hyperplasia, and the organ is firm and hard. In rare cases there is some adhesive peritonitis (perisplenitis).

Interstitial hepatitis may be present at birth, but may not cause obvious increase in the bulk of the organ. It is difficult to speak with any confidence of the proportion of cases in which any enlargement of the **liver** occurs, since it is not easy to prove that an apparent slight enlargement of the organ is real. The interstitial hepatitis may be attended by some atrophy of the hepatic cells and fibrous overgrowth in the portal canals. It may lead to jaundice, usually slight but occasionally intense, seldom or never to ascites, which, however, may occur in association with gummata in later childhood, a rare event.

Gummatous inflammation and sclerosis of the **intestinal** mucous membrane have been observed *post mortem* in infants dying soon after birth; but the clinical importance of such lesions is not great. Intractable diarrhœa occurs in many cases of syphilitic marasmus, to the production of which it no doubt contributes, but in such cases no lesion of the intestines is found beyond wasting of the intestinal mucous membrane.

Specific lesions of the **lungs** are observed in the bodies of children born dead or dying soon after birth; either fibroid induration with gummatous inflammation, or the "white pneumonia," in which patches of white hepatization are seen.

The **lymphatic glands**, unless the syphilitic eruption be complicated by suppuration, are, as a rule, little enlarged. Small shotty glands may be felt in many cases behind the ear, in the axilla, groins, or neck, and may persist for a long time.

The central **nervous system** is very seldom involved in infantile syphilis. Convulsions, retraction of the head, and opisthotonos have, on somewhat doubtful grounds, been attributed to syphilis. In

[1] Dr. Gee found it in 45 per cent., Dr. Coutts in 62 per cent., and in 19 per cent. in addition the organ was probably enlarged.

marasmic infants insomnia is sometimes a very prominent symptom. The infant is drowsy by day, but by night is restless, crying almost without ceasing, and sometimes screaming, as though in severe pain. At a somewhat later age pachymeningitis, cerebral sclerosis, and gummata may occur. Hemiplegia is in some few cases produced independently of the last-named lesion. Chronic hydrocephalus is, in rare cases, due to syphilitic disease of the membranes in the neighborhood of the fourth ventricle. The enlargement of the head is of the form usually observed in chronic hydrocephalus (see Chapter XLI.), but it seldom attains great proportions. The photograph of

Fig. 16.

Hydrocephalus in a syphilitic infant.

an infant (Fig. 16), in whom the enlargement was arrested while under the influence of mercury, shows the broad pear-shaped cranium, the flattened shallow orbits, and the depressed eyeballs. If the cranial bones are affected by the periosteal changes described below, the appearance may be extremely odd, as in the infant a photograph of whom is reproduced in Fig. 17. In this case the orbits were shallow, the sclerotics visible above the cornea, and the eyeballs depressed. The anterior fontanelle was large, and extended forward on to the forehead between two enormous frontal bosses, while the parietal bones were pushed outward and much thickened at their

upper edges. Children suffering from late syphilis are backward, and sometimes distinctly deficient in intellect.

Bone lesions, periosteal or epiphysial, occur in a very large proportion of all cases of inherited syphilis. According to Wagner, Birch-Hirschfeld, and other pathologists, epiphysial changes are present in all infants who die while suffering from the disease.

Osteoplastic periostitis producing a layer of porous osseous tissue occurs in the fœtus, and may be present at birth. The process may continue after birth, rendering the shafts thick, but true sub-periosteal nodes of the long bones, recognizable during life, are exceed-

FIG. 17.

Hydrocephalus in a syphilitic infant, showing bossing of cranial bones.

ingly rare in infants, and rare in older children. On the bones of the skull thickenings of periosteal origin are very common. They occur most often on the frontal and parietal bones, close to the anterior fontanelle, and not, as in rickets, on the frontal and parietal eminences. When large they form thick bosses on either side of the fontanelle, and if the thickening extends on to the forehead so that the site of the interfrontal suture is marked by a deep groove, the appropriateness of the descriptive term, "natiform skull," applied to the condition by Parrot, is very striking. *Post mortem* the thickened bone is found to be soft and porous, so that it can easily be cut with

a knife, and vascular. The margin of the boss may be well defined, or the larger part, or the whole of the bone or, indeed, of the skull, may be thick and vascular, the changes being greatest at the bosses. The lesion does not appear to cause pain, and the bones are little, if at all, tender. The bosses disappear usually about the end of the first year, and leave no trace, but are in many cases succeeded by rickety changes. In a few cases they have been known to suppurate and necrose. At a later age, seven years or older, a chronic gummatous periostitis may occur, especially of the tibia and palate, producing much thickening and deformity.

In **epiphysitis** the part affected is the proliferating layer at the junction of the diaphysis with the epiphysial cartilage. There is an excessive and irregular proliferation of the cartilage cells of the osteogenic layer, which undergo degeneration, and by interference with the vascular supply produce more or less extensive necrobiosis. The affected layer appears as an irregular yellow line, and the bony lamellæ of the adjoining part of the diaphysis are thin. If the destruction of tissue at the epiphysial line is sufficiently great the epiphysis may be detached, and when recovery ensues may become united to the shaft in a faulty position. In a few cases suppuration ensues upon the necrobiotic process at the epiphysial line, and then distinct grating may be perceived when the limb is handled. Suppuration is rare in infants except in the phalanges (dactylitis syphilitica). Epiphysitis, as has been said, may be present at birth, but it seldom produces discoverable swellings before the third month, more often a little later. The swelling is rather farther from the joint, and involves the end of the shaft more than in rickets, though it may be accompanied by effusion into the joint. The distal ends of the bones are more often affected than the proximal, the commonest sites being the lower end of the humerus, radius, ulna, femur, and tibia. The upper extremities are affected more often than the lower, and though the limbs on both sides may be attacked, showing a general symmetry, the lesion is commonly more severe in one bone than in others, or than in its fellow on the opposite side.

Associated with the epiphysial disease there may be complete loss of power in the affected limb, which lies or hangs flaccid and motionless in complete extension. At first there is some swelling of the limb, but later there may be a good deal of muscular wasting. As a rule, one limb only is affected, generally an upper limb, but occasionally two, and in rare cases all four limbs are attacked. Passive movement on handling of the limb causes pain, and it is customary to attribute the loss of power to the pain attending the epiphysial disease, and the wasting of the muscles to disuse, whence the term **pseudo-paralysis**. It is an early symptom, sometimes the earliest, and has occurred in the first week. The most usual age is

three or four months, but it has been seen as late as eight months. Distinct swelling about the epiphysial line may be absent, but tenderness is rarely or never wanting, though it may be slight and not distinctly limited to the neighborhood of the joints. Certainly in some cases it does not seem to be sufficient to account for the complete loss of power. Henoch doubts the connection, and the suggestion of Coutts that the paralysis and wasting may be due to peripheral neuritis is worthy of consideration. Recovery is usually rapid under treatment, but relapse has been known to occur.

The most different opinions have been held as to the **contagiousness** of inherited syphilis. Colles and Diday, for example, believed that it was more contagious and more virulent than the acquired disorder. Other writers of equal authority have held the opposite opinion. It is certain that instances in which syphilis can be proved to have been contracted from an infant suffering from the inherited disease are exceedingly rare; and it is possible that the contrary opinion may have been due to a failure to discriminate between acquired and inherited syphilis in infancy.

Late Syphilis.—Children who have suffered from syphilis in infancy are left in a condition of impaired health and nutrition, and are specially liable to succumb to some one of the many acute diseases, such as broncho-pneumonia or measles, to which their age is liable. A large proportion suffer from rickets in the second year. In some the normal rate of growth is checked, body and mind are stunted, and puberty delayed. At the age of ten to twelve years, or a few years earlier or later, signs of late syphilis appear in certain cases. The eye is the organ most often attacked, the commonest lesion being *interstitial keratitis*. One eye becomes tender, waters, and there is some photophobia; then the cornea becomes steamy and, finally, vascular, so that it has a pink-gray tint; gradually the pink color fades, the cloudiness clears away, and the cornea becomes almost or quite clear. Meanwhile the other eye has probably begun to pass through a similar series of changes, and for a time the vision may be no more than perception of light. Associated with the keratitis there may be iritis. *Choroiditis*, evidenced by patches of pigmentation and atrophy, may develop independently of keratitis. Sudden or gradual loss of hearing, without otitis and due probably to *labyrinthine disease*, ending in loss of hearing for the speaking voice, is occasionally produced. The *teeth* are liable to various lesions, but the characteristic deformity, described by Mr. Hutchinson, is a stunting of the upper central incisors (of the second dentition), which are peg-shaped, with a notch in the centre of the cutting edge. These three lesions, keratitis, deafness without otitis, and the peg-shape of the central incisors, form the "triad of Hutchinson." The other lesions occurring at this period are *gummatous* or *sclerosing* inflammations affect-

ing the bones, skin, throat and palate, brain and meninges, nose, liver, spleen, kidneys, testicles, lungs, and spinal cord.

Synovitis may occur under various forms.[1] In one fluid is effused very rapidly, generally into both knees. The affection may be mistaken for rheumatism, but the effusion is almost painless, and disappears quickly under antisyphilitic treatment. Effusion into joints may take place also as a complication of osteitis, and as a consequence of gummatous synovitis.

How many patients who have suffered from the early present also the latter manifestations cannot be stated, but it is certain that late syphilis is uncommon when compared with the frequency of inherited syphilis in infancy.

The **diagnosis** in a well-marked case of inherited syphilis in infancy can hardly be in doubt. In all obscure cases the history of the mother's pregnancies and the fate of other children of the family should be inquired into. A history of a series of abortions, or of children born dead or dying soon after birth, will alone excite legitimate suspicions, for a woman who has had children born dead owing to pelvic deformity will probably be aware of the fact, and hasten to communicate it.

The *acute coryza* of infants usually follows exposure, and is attended by rise of temperature, sneezing, and is often complicated by laryngitis or bronchitis; as has been said, doubt may arise if the case is first seen when the coryza is subsiding. Search should be made for any skin lesion, as, for instance, erythema of the soles, and branny desquamation about the eyebrows; and attention should be directed to the existence of anæmia, the sallow complexion of syphilis, or to enlargement of the bones. In the absence of any confirmatory symptoms, the physician will be well advised to keep his suspicions to himself, though it is often prudent to begin antisyphilitic treatment even before the appearance of distinct manifestations, which, however, will probably not be long delayed.

In young infants who suffer from syphilitic marasmus without other manifestations of the disease, the diagnosis must rest mainly upon the family history, and must be largely conjectural. At a later age a history of snuffles and rash may be obtained. The only symptom which is at all characteristic is nocturnal insomnia, and when this symptom is marked in a wasted infant who is judiciously fed, mercury ought not to be withheld. Even if the insomnia be due, as is sometimes the case, to the uric acid diathesis, it will probably be relieved by a judicious course of gray powder or calomel.

With regard to *rickets*, remembering the early age at which syphilitic bone changes, as compared with those due to rickets, commonly begin, the question in diagnosis is usually rather to recognize the

[1] H. B. Robinson, *Brit. Med. Journ.*, 1896, vol. i., p. 1191.

syphilitic basis of the rickets than to distinguish between two morbid processes which in the second and third years of life may be inextricably blended.[1] When the question arises it may be remembered that the swelling in syphilitic epiphysitis lies rather farther from the joint than in rickets, and in advanced cases the grating between the diaphysis and epiphysis may be detectable and decide the question in favor of syphilis.

Confusion can seldom arise between pseudo-paralysis and acute infantile paralysis. The age of the patient, the family history, the existence of syphilitic lesions of the skin, and the swelling of the epiphyses will usually suffice to prevent error.

Perlèche, an inflammatory disorder of the red margins of the lips, produces cracks at the corner of the mouth, but these are more acute and less deep than the rhagades of syphilis, and the affection occurs usually in epidemics in schools or institutions in which many children are brought together.

The **prognosis** in inherited syphilis is often at first uncertain. Setting aside pemphigus, it is safe to say that the prospects of recovery, so far as the skin lesions afford any indication, depend rather on the extent than on the nature of the lesions. At the same time, a very scanty eruption associated with much marasmus and wasting is of bad omen. The condition of nutrition is by far the most important element in prognosis, but even on this head a confident opinion cannot be formed until the effects of treatment have been observed. Sometimes even when emaciation is extreme the organism responds rapidly to mercury, and in a few weeks the infant increases extraordinarily in weight. If after a fortnight of systematic treatment the weight has not increased, the prognosis is bad, whatever the effect may have been on the skin. Marked enlargement of the liver and spleen is an unfavorable symptom, and the occurrence of jaundice is followed in almost all cases by death. The occurrence of pseudo-paralysis does not seem to aggravate the prognosis, and if the child survive the severity of the affection as it affects other organs, complete recovery in respect to the loss of power may be promised in those cases in which effective treatment can be applied.

In *late syphilis* the prognosis depends entirely on the extent and situations of the lesions. As a rule, recovery from interstitial keratitis is practically complete so far as vision is concerned, whereas the graver forms of retarded development with deficient intellect, upon which treatment has little or no effect, commonly leave the patient permanently crippled in mind and body.

Acquired syphilis in infants is identical in its manifestations with the acquired syphilis of adults. It is a less severe disease than inherited syphilis, to which it presents a general resemblance, but with

[1] *Conf.* Shattock, *Trans. Path. Soc.*, vol. xlii., p. 235.

certain differences. In the first place, there must be a primary lesion (chancre). Even if the case be in a later stage, evidence of its previous existence will probably be discoverable. This is an important point to bear in mind if the question of the transmission of syphilis by vaccination arise. In connection with the primary lesion there is considerable enlargement of the lymphatic glands. The first skin lesion is usually the roseola commonly seen in adults, and any subsequent eruption is usually scanty. The infant does not snuffle, or, at any rate, this is not an early and prominent symptom as in the inherited disease. On the other hand, the throat is often affected, and condylomata appear early, persist long, and are often luxuriant. The eruption has not the peculiar characters of that seen commonly in the inherited form, in particular the erythema of the feet and palms does not occur, the abdominal viscera are seldom enlarged, or only at a late stage, and the peculiar bone lesions are not met with.

The *prognosis* is decidedly better than in inherited syphilis, and turns mainly on the condition of nutrition and the effects of treatment on it.

In the **treatment** of syphilis in infants, whether inherited or acquired, but particularly in the former, the effects of mercury are most striking, especially the rapid and complete control which the drug has over the skin lesions of the early stage. It exercises also a very marked effect on the general nutrition, and under its use the anæmia diminishes rapidly. These beneficial results are, however, produced only after some months or more, and it is, therefore, necessary to continue the course of mercury for two months at least, and not to be induced by the disappearance of the rash to give up the remedy. When the first course is over the infant may be given tonics (the iodide of iron is specially recommended) for a month, and should then have another course of mercury for a month. This alternation should be practised three or four times, or till the end of eighteen months after the first manifestations. If symptoms appear later, the course should be repeated again and again if necessary, and if the child can be watched, anæmia or any failure in nutrition, at eight or nine years or at puberty, should suggest the propriety of submitting it again to specific treatment, since it is at these ages that the later manifestations are specially prone to begin. In infants it is often best to resort to inunction. The mother should be instructed to rub a scruple to half a drachm of blue ointment into the abdomen and back once or twice a day, and to cover the part with a soft handkerchief under the binder. In acquired syphilis, or if the services of the mother cannot be obtained, the ointment may be spread on the handkerchief, over which the binder is then somewhat firmly applied. A flannel binder should not be applied directly over the surface treated with ointment, as this practice is apt to cause an

amount of irritation of the skin which may render a suspension of the treatment necessary. Perchloride of mercury baths are the routine method of treatment adopted by some physicians. Baginsky, for example, states that since he began their use he has prescribed them in almost every case. The amount of mercury absorbed must be very small, but the treatment has the advantage that it disinfects the surface, and thus has a favorable influence on eruptions. A bath, which may be made by adding 1 pint of perchloride solution (1 in 1,000) to 3½ gals. water, should be given daily. For the internal administration of mercury no preparation is more convenient and satisfactory than gray powder; 1 gr. may be given twice a day, and if the dose is well borne it should be gradually increased until the infant is taking 2 gr. twice a day. It may be combined with compound chalk powder if vomiting is produced, or it may be replaced by perchloride, gr. $\frac{1}{60}$ to $\frac{1}{48}$ (liq. hydrarg. perchlor. (B. P.) ♏xv to xx) in flavored water thrice a day, or by calomel gr. $\frac{1}{12}$ twice a day. Mercury does not produce salivation in infants, in whom the functions of the salivary glands are very imperfectly established. It produces, however, after a time, diarrhœa, which should be an indication for stopping the drug for a time. In severe cases it may be desirable to give the drug both by inunction and internally. It is in such cases that hypodermic medication appears to be called for, but it is attended by considerable risk in very weakly children, and in stronger infants it is unnecessary to resort to a method of treatment which always causes a good deal of distress both to the patient and its guardians.

Eruptions on the buttocks should be treated by strict attention to cleanliness, by the use of antiseptic powders (calomel ʒss to starch powder ʒj, with or without a little zinc powder). White precipitate ointment (gr. xx to ʒj) is a good application for sores about the face, or calomel cream (calomel ʒj, olive oil ʒij, lanoline to ʒj) which is also very useful as an application to rhagades. Obstinate local lesions may be treated with oleate of mercury, 1 to 2 per cent., or with the red oxide of mercury ointment applied frequently in small quantity with a camel-hair brush. Condylomata are best treated by dusting with calomel and great attention to cleanliness. Iodoform may be made to alternate, as a dusting powder, with calomel, but in any case the application should be made several times a day, and the parts thoroughly washed beforehand.

The question of giving mercury to a mother suckling her syphilitic infant often arises. Chemical analysis of the milk has failed to reveal the presence of mercury, but very considerable improvement may follow in the infant on a course of mercury taken by its mother. This may be in part due to the improvement in her health due to the tonic action of the mercury, even in those cases in which she has not

been infected. If the mother presents any manifestations of syphilis there can, of course, be not the least hesitation in treating her, but it is not wise to rely upon this for the treatment of the infant. It should receive mercury itself by the mouth or by inunction.

Constant attention must be given to maintaining the nutrition of the infant. If the mother's milk is available, it should be prescribed to the exclusion of all artificial foods. If the snuffling prevent suckling, the milk must be drawn off and given with a spoon. Attention should be directed to the mother's digestion and general nutrition. Advantage will often be derived from giving her a tonic containing iron. If the child suffers from indigestion, small doses of pepsin or papain should be given after each feeding.

Pseudo-paralysis should be treated by keeping the limbs at rest, either by means of splints or by keeping the child on a pillow to which the limbs are secured by a broad bandage.

A child who has suffered from infantile syphilis should be carefully reared, warmly clad, well fed, and watched so that it can be put under treatment at the first evidence of any late manifestations.

In the treatment of late syphilis recourse must be had to iodides, either alone or alternately with short courses of mercury. The syrup of the iodide of iron is a useful remedy after pronounced symptoms have disappeared.

CHAPTER XV.

RHEUMATIC FEVER.

Etiology—Symptoms—Endocarditis and Pericarditis—Subcutaneous Nodules—Rashes—Diagnosis—Prognosis—Cervical Rheumatism—Treatment.

Rheumatic fever is a specific inflammatory process, affecting mainly serous membranes and fibrous tissues, to which individuals who inherit a certain type of nervous organization are peculiarly prone. It is sporadic in most, if not all, countries, is most prevalent in temperate climates in the spring, but presents epidemic increases at irregular intervals.[1]

Acute and subacute rheumatism present essentially the same features in children as in adults, but in them the affection of joints is often less marked and the disease less acute, though the liability to cardiac complications is probably greater. Under five years of age rheumatic fever is comparatively uncommon. It is more common between five and ten, but a larger number of first attacks occur during the second decade of life than in any other. Altogether, more than half the sufferers have their first attack before the age of twenty.

The most potent predisposing cause is inheritance from father or mother of a tendency to rheumatism, and the liability is greater if both parents are rheumatic.

The most frequent determining cause is chill, and its influence is increased by physical fatigue.

The most important manifestations of rheumatism are arthritis, endocarditis, and pericarditis, subcutaneous nodules, and erythema. To this list chorea should probably be added. Its relation to rheumatism is discussed below.

The characteristic affection of the joints in rheumatism is *synovitis*—acute or subacute—with serous effusion. There is injection of the synovial membrane, which may become covered with lymph. The fluid may be cloudy and contain shreds of fibrin, but the cellular element is scanty, only rarely giving the effusion a puriform appearance. The lesion of the pericardium is identical, but, owing to the

[1] According to Dr. Newsholme, epidemics follow periods of deficient rainfall, and prevail when the subsoil water is low and the earth-temperature at 4 feet is high. In these respects rheumatic fever would resemble summer diarrhœa, which is certainly an infective disease.

constant movement, the lymph effused is thrown into folds, or into small elevations causing a general coarse roughening of the surface. In the pleura similar effusion of lymph on the surface takes place, but the fluid effused is especially liable to become purulent.

The *onset* of acute or subacute rheumatism is generally sudden. The child complains of chilliness, of stiffness, is indisposed to move or eat, and perhaps vomits. The temperature is found to be elevated (101°–103° F.). The pain in the joints may be severe or, on the contrary, so slight that the child if in bed makes no complaint. There is, however, usually some tenderness, often very marked tenderness, though there may be little reddening of the skin and no fluid to be detected in the joint. The joints most often affected are the ankles, wrists, and knees, in the order mentioned; the hips and elbows more rarely. The metacarpal joints and the sheaths of the extensor tendons of the fingers appear to be more often affected than in adults. When the wrist joints and these tendons are simultaneously attacked the hand is kept in a rather characteristic attitude —the elbow is flexed, and the hand, slightly flexed at wrist and at the metacarpo-phalangeal joint, is supported by the other hand if it be unaffected, and carefully guarded from any jar or rough contact. As a rule, not more than one or two joints are attacked simultaneously, though many joints may be successively attacked. In a well-marked acute attack with high temperature, free perspiration having an acid odor is the rule, but in the less acute cases the sweating may not be very marked nor the odor noticeable. The very copious sweats so frequent in adults are certainly less common in children.

The most important characteristic of acute rheumatism in early life is the frequency with which the heart and pericardium are involved. As has been said, the pericardium in children behaves like a joint, and it may be the only joint, or at least the first joint attacked. In such cases the symptoms are far from characteristic. The child looks ill, has slight elevation of temperature, and if it complain of pain at all refers it to the præcordia or epigastrium. The diagnosis must then depend mainly on the recognition of the physical signs of pericarditis, which are discussed in another place. Endocarditis may develop independently of pericarditis, and even more insidiously, since there may be absolutely no pain. The onset can only be discovered by physical examination of the heart, which should be performed in every case in which rheumatism is suspected. It should be repeated systematically so long as the temperature remains elevated. The heart is affected in about three-fourths[1] of all

[1] Donkin found either old or active heart disease in sixty-one out of seventy cases, aged four to fourteen—*i. e.*, 87 per cent. Church (*St. Bart's Repts.*, vol. xxiii., p. 273) found cardiac affections in 83 per cent. of his cases under ten years, and 69 per cent. in the next decade; but the number of cases under ten was small.

the cases of acute rheumatism under fifteen. Pleurisy with effusion is a not uncommon complication, and in a few cases is the initial lesion. Dry pleurisy is a common malady in children who have suffered from acute or subacute rheumatism. Whether any large proportion of the cases of dry pleurisy or of pleurisy with effusion in children who have not suffered from rheumatism are rheumatic must remain doubtful; many are relieved but not cured by salicylates, in very much the same way as rheumatic arthritis. *Acute tonsillitis* may precede an attack of acute rheumatism; less often it comes on during its course, or as it is passing away. Rheumatic children are very liable to repeated attacks of acute tonsillitis; and it seems probable that some of those attacks frequently encountered in children who have not suffered from rheumatism are rheumatic in nature. Pneumonia is a not uncommon complication of rheumatism in children, but is probably so far accidental that it is not due directly to the rheumatic process. Rheumatic affections of the muscles may be the most pronounced feature of an attack, but, if they occur during the course of acute rheumatism, do not produce marked symptoms, owing to the child being bedridden. . Rheumatism of the sternomastoid may cause torticollis, and rheumatism of the abdominal muscles acute abdominal pains and tenderness so severe as to simulate peritonitis.

Subcutaneous nodules are an interesting form of rheumatic inflammation of the fibrous tissues. They are of considerable diagnostic importance, since they may be present in obscure rheumatic affection of the heart, even when the joints are not obviously involved. Usually, however, when they are present the joints are affected. They are said by Coutts[1] to be discoverable in 20 per cent. of all cases of acute rheumatism with heart disease in children. When they are associated with heart disease the cardiac affection is often severe and progressive. Though they may appear during a period of pyrexia, their development does not seem necessarily to be attended by fever. In size they vary from a mass barely perceptible to the touch to that of an almond; but usually they are not larger than a melon seed. They are movable under the skin. There may be few or many. They develop rapidly, sometimes appearing in successive crops, and may disappear in a week or ten days, which is rare, or persist for several months. They are little, if at all, tender. They occur usually about the joints, especially the elbows, the knuckles, the malleoli, at the edge of the patella, and sometimes over the vertebral spines, the scapulæ, the iliac crest, and the occiput. It is possible that similar nodules may be produced by syphilis, but with this exception, if it be one, they are found only as a rheumatic manifestation.

[1] Donkins's "Diseases of Childhood (Medical)," London, 1893, p. 213.

Various **rashes** may be observed. Sudamina are very common, and a fine red rash resembling the early stage of the scarlatina exanthem is not uncommon. Erythema, of various forms, may accompany, precede, or follow the arthritic attacks. Urticaria occurs occasionally. Purpura sometimes ensues on intense erythema, but it may develop rapidly during acute rheumatism, and may be attended by hæmaturia.

Erythema nodosum is certainly more common in rheumatic children than in others, though some authorities doubt whether in truth it is a rheumatic affection. After a fever articular pains in the lower limbs and general malaise have existed for several days, oval symmetrical swellings appear over the tibiæ. They are of a bright red color and tender; their long diameter, which is vertical, measures from 1 to 2 or 3 inches. After a day or two the swelling, at first tense, becomes soft and of a dusky hue. The color then passes through the stages usual in a bruise, and the lesion disappears in eight or nine days. There may be several symmetrical swellings over the tibiæ, or successive crops may come out. More rarely, erythema nodosum appears on the outer side of the leg or on the arms. The attack is not always accompanied by arthritis, but, on the other hand, in some of these non-arthritic cases endocarditis ensues. Taking all cases of erythema nodosum, it is found that acute or subacute arthritis or cardiac lesion occurs in the majority, and that in some of the minority there is a family history of rheumatism.

The **general symptoms** of rheumatism vary very greatly in intensity, and are in proportion, as a rule, to the amount of fever. The child feels and looks ill, and even when there is little or no pain is disinclined to move. The bowels are constipated at first, food is refused, but drink eagerly swallowed. The temperature presents great variations, but is, on the whole, lower than in adults. In an ordinary attack it will range between 101° and 103° F., in subacute cases it may not rise much above 100° F., and serious cardiac lesions may be produced without any observed elevation of temperature.

Hyperpyrexia is rare in children.[1] The symptoms are a sudden rise of temperature to 106°–110° F., delirium or coma, headache, pain in the back, twitching of the face and fingers, and hurried respiration and pulse. These symptoms were formerly attributed to meningitis. As a matter of fact, meningitis, whether cerebral or spinal, is not, it would appear, produced by the rheumatic poison.

The blood is rapidly and seriously affected in acute rheumatism. There is marked leucocytosis, and a profound degree of anæmia may

[1] The report of the Committee of the Clinical Society on Hyperpyrexia (*Transactions*, vol. xv., p. 265) dealt with 1,300 cases of rheumatism, but only 1.8 per cent. were under ten years. None of them suffered from hyperpyrexia. In the next decade, however (ten to twenty), there were a larger number of cases (34.6 per cent.), but they yielded only 19.4 per cent. of the hyperpyrexial cases.

be brought about very quickly. The rapid formation of a fibrinous clot in the right ventricle or pulmonary artery is an occasional cause of a sudden fatal termination during the course of an acute attack, or even after convalescence has, apparently, become established. Subacute rheumatism also may entail extreme anæmia, and the characteristic appearance which patients who are liable to attacks of subacute rheumatism present is due to this cause, though in addition the skin has a slight waxy or sub-icteric tint.

The *diagnosis* of rheumatism, whether acute or subacute, if it involve the joints is commonly not difficult if the case can be observed for a few days. In the first place, rheumatism is the most common cause of acute arthritis in children, and the subsidence of inflammation in one joint with its appearance in another is extremely characteristic. Multiple arthritis secondary to exanthematous diseases (*q. v.*) is seldom so acute or so well-marked as to lead to error, though in the absence of a history some difficulty may arise. In this connection diphtheria must be thought of, since pains in and about the joints are of not uncommon occurrence in that disease, and the local affection may be so mild as to provoke no marked symptoms referable to the throat. The arthritis secondary to gonorrhœa is exceedingly rare in children. Pyæmic arthritis from other causes is also rare, but may simulate acute rheumatic arthritis very nearly; the local inflammation and reddening of the skin is greater, subsidence in the joints first affected is, as a rule, less rapid and complete, and the temperature will commonly show marked pyæmic characters. In the absence of distinct evidence of a source of pyæmic infection, the diagnosis is difficult and is probably seldom made in the early stage. The possibility that the arthritis is due to acute epiphysitis (*q. v.*) must also be borne in mind, especially in young children and infants.

Sanguineous effusion into the joints in hæmophilia may be attended with pain and some general disturbance, so that in the absence of a definite history some hesitation may be felt at first. The pain, however, is slight, compared with that attending rheumatic synovitis which has produced like distension of the joint, and in a few days the true nature of the case will be made clear. Scurvy-rickets is hardly likely to be confounded with rheumatism if the ages at which the two affections occur are borne in mind, more especially as the tender swellings produced in scurvy are often seated over the long bones, or, if in relation with the joints, do not cause effusion into them in the early stage. In like manner a careful physical examination, and a consideration of the age and the surrounding circumstances of the patient, will prevent the tenderness and epiphysial swelling of acute rickets from being supposed to be rheumatic synovitis. It should be remembered that infantile paraly-

sis may be accompanied at its onset by flying pains in the limbs and by some tenderness of the joints, especially of the affected limb.

[We would emphasize the importance of the conception of rheumatism in children as set forth by Cheadle and other English writers. Unless we realize and accept this conception of the disease, it will often escape us. If we look, in children, only for the clinical picture as seen in adults, the sudden onset with high temperature, marked joint symptoms and acid sweats, we shall rarely find rheumatism. This picture is seldom seen. We must look for other and more varied manifestations, occurring more irregularly, not grouped within a few weeks or months but extending over a period, it may be of years. Especially must we be on the lookout for endo- and pericarditis; one is rarely present without the other in the rheumatic heart affections of children. These are much more common than the joint symptoms. "Endocarditis is at its maximum, arthritis at its minimum."

The occurrence of the subcutaneous fibrous nodules is not as common in America as it apparently is in England. But two cases presenting these nodules have been seen in seven years' experience with two large Chicago clinics. With a child who has had, at any time in his life, attacks of tonsillitis, or of erythema, or of bronchitis, who gives a history of indefinite joint pains, or in whom subcutaneous nodules have been noted, who in any way presents one or more of the manifestations of rheumatism, the heart must be watched most carefully with the recognition that these apparently widely different phenomena are in reality closely related, and but symptoms of a single underlying diathesis.]

The **prognosis** of acute rheumatism is in children somewhat better than in adults, so far as recovery from the acute attack is concerned. Death is brought about most often by pleurisy or pneumonia, rarely by pericarditis or endocarditis, at least in a first attack. If the heart has been damaged by previous attacks, death may be caused by cardiac failure. On the other hand, a favorable prognosis as to the remoter future must be given with the utmost caution, even in cases of subacute rheumatism. The great frequency with which the heart is involved in children has already been mentioned, and there is no doubt that a child who has once suffered from rheumatism, acute or subacute, is extremely liable to fresh attacks, during one of which the heart is very likely to be involved.

Cervical rheumatism is a manifestation of rheumatism sufficiently common in children, and sufficiently characteristic to deserve special mention. The rheumatic process may attack the articulations of the cervical vertebræ, their ligaments, or the muscles. The child is seized suddenly by severe pain in the neck, which is held rigidly. There is tenderness along the spine. Frequently, owing either to

simultaneous affection of the muscles or to their contraction to protect the painful part, there is torticollis, or retraction of the head. The pain is very much increased by any movement of the head. The attack may be the initial symptom of acute rheumatism which subsequently runs an ordinary course, or it may occur as an isolated phenomenon. Occasionally it is complicated by endocarditis or pericarditis. The course of the affection is usually subacute, and recovery ensues; but in some cases chronic arthritis or fibrous thickening of the ligaments and muscles remains, producing lasting rigidity or distortion of the neck. The only difficulty in *diagnosis* is to distinguish the affection from tuberculous osteitis of the cervical vertebræ; the sudden onset and the severity of the symptoms at an early stage will generally prevent error, and as a rule the effect of treatment by sodium salicylate will remove any uncertainty. In many cases, however, this drug, though it leads to improvement, fails to effect a cure, and in such cases careful massage of the parts is to be recommended. Recovery may often be hastened by mild counter-irritation.

The **treatment** of acute rheumatism in childhood must follow the same lines as in adults. The patient should be put to bed in a flannel nightgown between blankets or flannel sheets. The joints should be enveloped in cotton-wool, and a cotton-wool or flannel pad lightly but firmly bandaged over the chest by a many-tailed bandage. Great relief may be obtained by the application of suitable splints when the knees, elbows, or wrists are involved. Local applications may be tried if the pain be severe—chloroform liniment, aconite liniment, or the chloroformum aconiti, B.P.C. Osler recommends hot cloths saturated with Fuller's lotion (see Appendix).

[Carbonate Sodium,	℥vj
Laudanum,	℥j
Glycerine,	℥ij
Water,	℥ix]

In Germany cold compresses or ice-bags are much used. Small blisters above and below the joint, or, in the case of the knee, along the outer and inner sides of the patella, are valuable means of relieving pain in subacute cases, but are not to be recommended in acute attacks or in young children. When the ankles or wrists and fingers alone are involved relief may be obtained by local hot baths at about 100° F.[1] They should be repeated two or three times a day, the parts well dried and then wrapped in cotton-wool.

Of internal remedies the salicylates take the first place. Under their influence the pain is nearly always relieved if not entirely removed, and although they do not prevent heart complications, they

[1] Lenhartz (Penzoldt and Stintzing's "Handbuch," Bd. v., s. 159) recommends the addition of common salt, ½ to 1 lb. in a wash-handbasin of water.

perhaps render the attack shorter, and certainly easier to be borne. In a disease which tends to produce exhaustion this is an advantage not lightly to be set aside.

The amount of sodium salicylate which may be given to a child five or six years old, during the first two or three days, may be set down at 60 to 80 grains. It should be given in divided doses every two or three hours, day and night.

[Sodii Salicylatis, ℥j
Tn. Aurantii Rec., Glycerini, āā ℥ij
Aq., ad ℥j
Dose—℥j every 2 or 3 hours.

Sodii Salicyl., ℥j–℥ij
Liq. Ammon. Acetat., Syr. Aurantii, āā ℥iij
Aq., ad ℥j
Dose—℥j every 2 or 3 hours.
Appendix.]

As the pain subsides the daily quantity should be reduced, but the smaller doses should still be given at frequent intervals. This seems to be important, since the drug is rapidly eliminated. Salol, which is decomposed by the alkaline intestinal secretions into salicylic acid and phenol, yielding about 60 per cent. of the former, has been recommended, partly with the object of insuring the continuous absorption of a salicylate. It may be given in powder. Salophen, which under similar conditions yields 51 per cent. of salicylic acid, has been preferred when it is desired to keep up the action of salicylic acid for long periods, since it is less poisonous and more slowly decomposed than salol. Salicin does not appear to have any advantage over salicylate of sodium, and has the disadvantage of being less soluble. The dose for a child of six is about gr. iij every three or four hours. That the salicyl compounds do not prevent the heart being attacked is admitted, and it has been asserted that, so far from preventing, they rather favor relapses. On the other hand, it is held by some, as I believe correctly, that this opinion is due to the practice of stopping the drug so soon as the joint pains have been relieved. This view is maintained especially by French physicians.[1] Jules Simon begins with a small dose, ½ gramme (7½ grains) on the first day, and increases it by ½ gramme a day until the maximum dose, for a child of ten, of 3 grammes (45 grains) is reached. He then begins to reduce it by 15 grains daily until the child is taking only 15 grains a day; this dose is continued for a week at least. The whole period of treatment occupies a month or more, and the child is kept in bed for that period whether it present rheumatic pains or not.

In some few cases the salicylic compounds appear to exercise no influence over the disease, and in other cases, rarer in children than in adults, they produce toxic symptoms, delirium, vomiting, epistaxis,

[1] See the "Year-Book of Treatment" for 1895, p. 194; 1896, p. 161.

intestinal hæmorrhage, dyspnœa, or great cardiac weakness.[1] When the remedy has to be stopped for one of these reasons, the best alternative treatment is the alkaline. It is founded on the theory that the symptoms of rhumatism are due to an excessive production of acid (it is said, lactic acid). The object aimed at is to render the urine alkaline. The drawback to the treatment, which undoubtedly relieves the joint pains and, it is said, diminishes the liability to cardiac complications, is that it has a very depressing effect, and increases the tendency to anæmia. It is worse than useless to give insufficient doses of alkali for a long period. The dose should be regulated so as to render the urine alkaline; at least 30 to 40 grains must be given to a child of ten every three or four hours. It may be combined with potassium acetate, as advised by Fuller (see Appendix).

[Sodii Bicarbon.,	gr. xxx to xl
Potassii Acetatis,	gr. x
Aq.,	℥ss

In effervescence with citric acid (gr. x) or fresh lemon juice (℥jss) every 4 hours, to be reduced after 24 hours.]

The desired effect on the urine should be obtained within twenty-four hours, and the amount of alkali then diminished but maintained at such a quantity as will just keep the urine alkaline. Quinine may be combined with the alkali to diminish the depressing effect.

[Quinine and Alkali (Garrod).

Quininæ Sulph.,	gr. ij
Potassii Bicarb.,	gr. xx
Tn. Aurantii,	♏xij
Mucil. Acac.,	℥ss
Aq.,	ad ℥ij

(A single dose.) The quinine is rubbed up with the bicarbonate, dissolved in water and the mucilage added afterwards. Appendix.]

When the joint pains are severe, small doses of opium (by preference, perhaps, Dover's powder, gr. v to a child of ten) may be given on the first night; but it will seldom be necessary to repeat the dose, if salicylate can be taken. The use of opium, absolute rest in bed in a darkened room, and cotton-wool wraps to the joints were the main points in the so-called "expectant treatment" of Gull and Sutton.[2]

An alkali is by some physicians combined with sodium salicylate in the treatment of both acute and subacute rheumatism.

[1] I am not aware of any instance in which it has produced albuminuria in a child. Probably the toxic symptoms are not infrequently due to impurities in the "artificial" salicylate. The "natural" product is therefore to be preferred.

[2] See the admirable article in Fagge and Pye-Smith's "Medicine," vol. ii., p. 702, 3d edit. The whole article deserves careful perusal. The dose of opium was, for an adult, 1 grain nightly, or oftener if pain were severe.

[Sodii Salicylatis,
Sodii Bicarbonatis, āā ℥jss
Tn. Aurantii Rec.,
Glyc., āā ℥iij
Aq., ad ℥ij
Dose—℥ij every 3 or 4 hours. Appendix.]

But this line of treatment, if it be recommended at all, seems better adapted to subacute recurrent attacks. Such attacks are often little amenable to the salicylate treatment, and when rheumatic cachexia with marked anæmia has become established, it should not be resorted to. In such cases iron should be given in the form of the solution of the perchloride, or, if that drug produce gastric disturbance and diarrhœa, as is sometimes the case, it may be replaced by the citrate of iron and ammonia (gr. v thrice daily to a child of eight). When stiffness, aching, or flying pains in the joints are troublesome, the addition of 3 to 5 grains of sodium salicylate to each dose of citrate sometimes relieves; but tincture of colchicum in doses of ♏x to xv thrice a day (for a child of ten) will commonly be found a more effective remedy. With the colchicum may be combined small doses of potassium iodide (gr. v thrice daily for a child of ten), and the colchicum should after a few days be replaced in this combination by a grain of quinine. Drugs, however, with the exception, perhaps, of iron, commonly fail to exercise any conspicuous effect, and the greater reliance must be placed on attention to diet and clothing.

CHAPTER XVI.

CHRONIC RHEUMATIC AFFECTIONS.

The Rheumatic Cachexia, and Chronic Rheumatism—Rheumatoid Arthritis.

THE *nomenclature of diseases* enumerates in succession in the list of general diseases :—Rheumatic fever (acute rheumatism), rheumatism (subacute and chronic rheumatism), gout, and osteo-arthritis (rheumatoid arthritis). To these must be added the various forms of arthritis, especially polyarthritis, which occur as complications of acute infectious diseases, and chorea, which has certainly intimate relations with rheumatism. The group is a somewhat miscellaneous assemblage, but the arrangement is convenient from the clinical point of view, since it brings into relation morbid states which must in some cases be compared and in others contrasted.

Rheumatic Cachexia.—A child which has once suffered from acute or subacute rheumatism is, as had already been said, very liable to suffer renewed attacks; and children who have had chorea are likewise peculiarly liable to suffer from recurrent attacks of rheumatism. Children who present the rheumatic diathesis to a well-marked degree, even though they have never had an attack of definite rheumatism, are very apt to pass into a condition of debility characterized by anæmia and recurrent rheumatic pains in the joints and muscles, with, perhaps, frequent tonsillitis. To this condition the term *rheumatic cachexia* may justly be applied. It occurs chiefly in girls shortly before menstruation, and in boys a few years before puberty. The child often grows quickly, "outgrows its strength," as it is said. Its appetite is capricious; it is very easily fatigued, and slight exposure, if combined with fatigue, is almost certain to be followed by sore throat (tonsillitis) or rheumatic pains, or by subacute or acute rheumatism. Even if an acute attack be escaped, it is rare for the heart not to be affected sooner or later, and it is cases of this type, which yield, I believe, the larger proportion of the cases of malignant endocarditis. It is to such cases that the term *chronic rheumatism* is most properly applied; but the term has been so much abused that it is better to avoid it, more especially as such children are, as has been said, very liable to acute or subacute attacks.

Change of air, often regarded as a panacea for all conditions of chronic ill-health, little avails the sufferers from the rheumatic

cachexia. They often experience their most serious attacks on returning to a town after a country holiday. When the place of residence can be selected, the warm relaxing climates sometimes chosen should be avoided. A dry inland place on high ground away from river or lakes, and with few wet days, probably offers the best prospect for these patients, owing mainly, no doubt, to the fact that in such localities they are able to get out of doors on most days of the year. Neither the seaside nor mountainous districts suit them. Attention should be directed to the sanitary arrangements of the house, its warming, lighting, and drainage. Contamination of the air of the house by emanations from sewers certainly produces a deterioration of the general health, and not improbably has an even more direct influence in determining tonsillitis and other rheumatic manifestations.

Rheumatoid arthritis (arthritis deformans, osteo-arthritis) occurs in childhood somewhat more frequently than appears to be generally recognized. Following Charcot's division of the cases of this disorder into (*a*) Heberden's nodes; (*b*) the general, progressive polyarthritic form; and (*c*) the monarthritic form, it may be said that the last is extremely rare, if indeed it ever occurs in childhood.[1]

Heberden's nodes, small nodules which form generally at the distal extremity of the second phalanges of one or more fingers, are not very uncommon. Their appearance, or increase in size, is sometimes accompanied by pain, redness and swelling of the joints of the fingers; this passes away, leaving the nodules in a condition of quiescence unattended by pain, except when the part is knocked.

Progressive polyarthritis deformans commonly develops in children in a manner which certainly presents considerable clinical resemblance to a mild attack of rheumatic fever, although the pathology is probably different. There is some elevation of temperature, pain, tenderness, and swelling; sometimes redness of several, often of many joints. The symptoms are little, if at all, controlled by salicylates, but subside in a few days; another attack occurs after a short interval, and after a time it is perceived that the ends of the bones are enlarged, and the movements of the articulations limited. The form of the joints becomes gradually distorted, owing in part to the formation of osteophytes, and in part to thickening of the ligaments. The synovial cartilages disappear, and are replaced by an ivory-like thickening of the ends of the bones. The muscles moving the affected joints become atrophied to a greater or less degree, and the hands, wrist, and limbs assume various abnormal positions. The course of the disease is, on the whole, progressive; but after the subacute exacerbations considerable improvement may occur, and the disease may remain quiescent for years.

[1] Marfan, "Traité des Mal. de l'Enf." (Grancher, Comby, et Marfan) denies that it occurs, and I have never seen a case.

In some cases the distribution both of the osseous and articular lesions and of the muscular atrophy is remarkably symmetrical, but "glossy skin," and other skin changes are at least uncommon in children.

The **etiology** is obscure. In the most typical cases the distribution of the lesions undoubtedly suggests a central nervous origin, and mental anxiety and other depressing emotions sometimes appear to be determining causes of an attack or exacerbation in children as in adults. In some cases there is a family history of joint affections commonly described as rheumatic. The disease is very uncommon under five years, rare under ten. The symptoms are aggravated by exposure to cold and damp. It is usually taught that the condition has no relation to rheumatism. Even acute rheumatism is, however, held by many to be due to a primary affection of the nervous centres, and there are cases of chronic rheumatism so-called, with heart lesion, which in other respects present a general resemblance to the polyarthritic form of rheumatoid arthritis. The diagnosis is, therefore, sometimes difficult, especially in children who have suffered from several febrile attacks with joint pains, attacks always spoken of by parents as rheumatic fever or rheumatism. In well-established cases in which the characteristic deformities about the joints and atrophy of muscles exist, the diagnosis is usually easy. Periosteal nodes, a rare complication of acute or subacute rheumatism, may give rise to a superficial resemblance, but the nodes, as a rule, disappear rapidly under salicylates.

The treatment during the exacerbations should consist of rest in bed, careful dieting, and soothing applications to the joints, which should be wrapped in cotton-wool. Salicylates do not, as a rule, exercise any influence. In the intervals the general nutrition should be improved by every available means, including careful dieting, warm clothing, and change of air at suitable times. Cod-liver oil and malt, the milder preparations of iron, and arsenic are valuable adjuvants, and advantage is often derived from strychnine in doses as full as can be borne. Galvanism yields good results in some cases. One pole should be placed in a basin of salted water, while the other electrode is placed on the spine over the cervical or lumbar enlargement, as the case may be. The hands or feet are then placed in the basin, and the current passed at first with the lower pole negative, and subsequently reversed.

CHAPTER XVII.

INFECTIVE ARTHRITIS.

Polyarthritis and Monarthritis — Scarlet Fever — Diphtheria — Typhoid Fever — Mumps—Gonorrhœa—Acute Epiphysitis—Prophylaxis and Treatment of Infective Arthritis.

Not only gonorrhœa, and other purulent infections, but also scarlet fever, typhoid fever, cholera, mumps, diphtheria, erysipelas and other specific infectious fevers may be complicated by arthritis. The arthritis may be due either to the action of the specific infection or to a secondary infection by pyococci. In the former alternative the smaller joints are those most often affected, many being attacked simultaneously or in rapid succession. The affection is a polyarthritis, and thus resembles acute rheumatism; but it is commonly mild and transitory. In the latter, on the contrary, the large joints are those usually affected, and the inflammation is often limited to one, but it may be so severe as to cause more or less complete disorganization of the articulation attacked.

Polyarthritis is a rare complication of any of the infectious fevers. Scarlet fever is held to be that most often thus complicated. This opinion is perhaps due to the fact that the arthritis which occurs as a complication of scarlet fever is commonly more severe than that observed in other fevers, and that it is sometimes attended by endocarditis.

In those cases in which many joints are attacked in succession, all the structures of the articulation affected are involved; but there may or may not be sufficient effusion to distend the joint. The effusion is serous, and in some cases there is teno-synovitis. The symptoms are usually characteristic—pain in the joint increased by movement, tenderness, more or less reddening of the skin, and swelling of the joint. The pain and tenderness are less severe than in acute rheumatism, and the whole affection is milder.

Arthritis due to pyococcal infection appears to commence as a catarrhal synovitis, but at an early stage the cartilages and ligaments are involved, and the effusion becomes sero-purulent, or purulent. The affected joint is painful and tender, and the skin hot and red. The course of the arthritis varies; in some, perhaps the majority of cases, the symptoms subside rapidly, and the functions of the joint

are restored. In others ankylosis ensues with atrophy of the muscles about the joint. When suppuration occurs, and separation of the epiphyses, dislocation may ensue, with more or less complete disorganization of the joint. In such cases there is a general infection, often of distinctly pyæmic type.

Polyarthritis when it occurs as a complication of *scarlet fever* comes on usually rather late; that is, during the third week after the onset of the disease. It is believed to be less frequent in children than in adults. It affects by preference the smaller joints, those of the hand, wrist, and foot, less often the ankle, sometimes those of the cervical vertebræ, producing retraction of the head or flexion on one or other shoulder. The joints are not much swollen, the skin is little reddened, the pain is not severe. As a rule recovery is rapid and complete, but occasionally ankylosis occurs. Suppurative arthritis is a rare complication of scarlet fever: it occurs almost exclusively in severe cases presenting other pyæmic symptoms; it is generally limited to one, or to few joints.

Arthritis is an occasional complication of *diphtheria;* it comes on usually in the second or third week, and the articulations most often affected are the knees and other large joints. Pain is usually out of proportion to the visible swelling or other evidence of inflammation. Suppurative arthritis due to secondary infection is a rare accident. Mild attacks are perhaps rather more common in cases treated by the antitoxic serum than in others.

Arthritis affecting usually many joints to a varying but, in most instances, slight degree, is a rare complication of *typhoid fever*. It is observed in the second or third week. Even in the absence of much or any swelling of the joint, pain may be severe, but is usually of short duration. Another form of arthritis occurs at a later date; it is usually limited to one joint, generally the hip, and occasionally results in ankylosis or dislocation. In some cases, apparently rheumatic, the lesion is in reality due to osteo-myelitis, attended by effusion, which may be purulent, into the joint. In other cases the osteo-myelitis is of a more chronic type, and may lead to the formation of exostoses. "Typhoid spine," a condition in which all movements of the spine are painful, is occasionally met with in children.

Mumps is in very rare instances complicated by arthritis or tenosynovitis of mild type and short duration. In certain epidemics of *cerebro-spinal meningitis* polyarthritis is observed in a large proportion of cases.

Gonorrhœal arthritis may occur in children, especially girls, in whom gonorrhœal vulvo-vaginitis is not very uncommon; it has also occurred as a complication of gonorrhœal ophthalmia in new-born children. The onset of the arthritis is attended by general febrile

symptoms, and as they subside it is found that one or more joints are hot, swollen, and painful. The joint most often attacked is the knee; then the wrist, ankle, the small joints of the hands and foot, least often the hip-joint. As a rule complete recovery takes place, even though the fluid effused into the joint has been purulent, but obvious improvement may not be observable for several weeks. An occasional complication is atrophy of the muscles about the affected joint.

Acute epiphysitis, that is to say, acute osteitis of the epiphysis or of the diaphysis near the growing line is a not uncommon affection in young children. It produces acute local pain and tenderness, and fever of varying intensity. It attacks most often the hip, elbow, shoulder, and ankle, and is commonly limited to one joint. In acute cases the intensity of the local process, and the fact that the swelling in the early stage is distinctly away from the joint (except in the hip) will assist the diagnosis from acute rheumatism, but mistakes have been made by the most skilful. A form of this affection which occurs in infants has been specially described under the name *Acute Epiphysitis* (or *Arthritis*) *of Infants*. Most of the cases occur in infants under one year, and may develop a few days after birth. The acute inflammation at or near the ossifying centre leads to necrosis and suppuration. The abscess, in most cases, opens into the joint, and produces an acute arthritis attended by much local swelling, tenderness, and reddening of the skin. After a few days of fretfulness it is noticed that the infant does not move the limb, and that passive movement causes acute pain. The hip is most often attacked, and next the knee. Other joints may also become inflamed, and the condition of those earliest attacked may improve, but after a short time the more serious affection of one joint becomes evident. The disease is certainly pyæmic, and the secondary affection of other joints, when not due to direct extension of the osteo-myelitis, is of this nature. The prognosis is extremely bad, nearly half the cases dying of pyæmia. Whenever there is reason to suspect this condition the limb should be kept at rest by bandaging it to a splint, or in very young infants by bandaging the child to a pillow so as to prevent movement of the affected limb. Owing to the fact that rheumatism is extremely rare in infants, if indeed it ever occur, a mistake in diagnosis, in spite of the great resemblance between the two diseases in an early stage, is little likely to be made. It is safer to assume that a case of multiple arthritis, or of multiple inflammatory affection about the joints in infants is pyæmic, and to watch for the earliest indications which may point to the formation of pus in the neighborhood of the joint, or of infusion into it. Early incision and drainage appears to hold out the best prospect of recovery in cases in which the disease makes progress in spite of keeping the limbs at rest.

Prophylaxis.—The occurrence of secondary arthritis in so many forms of acute infectious disease is an additional proof of the importance of the prevention or early treatment of all suppurative complications such as those occurring in the mouth, throat, ear, conjunctivæ, or vulva. The *treatment* of purulent arthritis must be conducted on general surgical principles, but it is desirable that the joint should not be kept immobile longer than is necessary to relieve pain, and that if wasting of muscles occur massage and galvanization should be resorted to at an early date. In those forms of secondary arthritis which occur earlier in the course of the specific infection, by which probably they are, at least in some cases, directly produced, the ordinary treatment of rheumatism has little influence. Sodium salicylate has not the marked effect customarily observed in true acute articular rheumatism. It does, however, exercise some influence and may be used in combination with antipyrin, or these drugs may be replaced by quinine in cases in which a depressing effect is to be feared from antipyrin. The patient should be kept in bed and the affected part wrapped in cotton-wool.

In gonorrhœal arthritis the most effective means should be at once taken for the cure of the local infection, and they should be persevered in so long as there is any evidence of local inflammation (vulvitis, conjunctivitis).

CHAPTER XVIII.

CHOREA.

General Characters—Etiology—Pathology—Symptoms—Recurrence—Treatment.

CHOREA MINOR (ST. VITUS'S DANCE).

St. Vitus's Dance, called chorea minor to distinguish it from a form of hysteria to which the term chorea major has by misfortune been given, is a common disease of growing girls, and is far from uncommon in boys. It has been well said by Sturges [1] that chorea consists in an exaggerated fidgetiness. It is an extravagant exaltation of that continual unrest which is a natural characteristic of childhood. Its movements, that is to say, resemble those due to emotion, the same muscles being affected in the same kind of way. Consistently with this comparison, the muscles of the upper part of the body are much more often affected than the rest, and the hands suffer most of all. The disease is also sometimes spoken of as "Sydenham's Chorea" since he was the first writer to give any accurate description of it. He says [2] "This is a kind of convulsion which attacks boys and girls from the tenth year to the time of puberty. It first shows itself by limping or unsteadiness in one of the legs, which the patient drags. The hand cannot be steady for a moment. It passes from one position to another by a convulsive movement, however much the patient may strive to the contrary. Before he can raise a cup to his lips he makes as many gesticulations as a mountebank, since he does not move it in a straight line, but has his hand drawn aside by spasms, until by some good fortune he brings it at last to his mouth. He then gulps it off at once, so suddenly and so greedily as to look as if he were trying to amuse the lookers-on."

Etiology.—Girls suffer from chorea more than boys, in the proportion of about two to one, and about three-fourths of the cases occur between the ages of 5 and 15 years. The disease is rather more common among the children of the poorer classes, and is more prevalent in certain localities and among certain races than in others. It ap-

[1] "On Chorea, or St. Vitus's Dance, in Children" (Second Edition. London: John Bale & Sons, 1893).

[2] "The Works of Thomas Sydenham" (London: The Sydenham Society, 1850), vol. ii., Processus Integri, xvi.

pears, for instance, to be relatively rare among negroes in the United States, where white children suffer to about the same extent as in Great Britain. More cases occur in spring, and, on the whole, the seasonal incidence of the disease corresponds very closely with that of rheumatism. [Townsend assigns as an important cause of the more frequent occurrence of chorea at this season the mental exertion and depression arising at the end of the school year. He notices an increase in the number of cases in October also, cases especially predisposed to the disease and needing only the excitement and mental exertion of a month of school to precipitate the attack.] The subjects of chorea are generally bright, excitable children, and their history shows frequently the existence of some family predisposition; it is not uncommon to find that the mother or a brother or a sister has suffered from the disease. Psychical disturbances and emotional upset caused by fright, scolding, sudden grief, or injudicious religious excitement appear to be common determining causes of an attack, which may develop even within a few hours. "Over-pressure" at school is a cause frequently assigned, but it operates indirectly by causing a condition of emotional excitement, just as in adults nervous breakdown is commonly due rather to anxiety than to overwork. The excitement produced by an approaching examination, or the worry due to the unreasonable demands of an unskilful teacher, rather than the actual number of hours the child works, are the important points. Imitation has been considered as an important determining cause, but many of the instances quoted appear to belong to the category of hysteria rather than of chorea; when imitation is operative it acts probably through the emotional shock caused by seeing a relative or companion reduced to the distressing condition which a well-marked attack of chorea produces. A history of some accident or injury, sometimes of a surgical operation, is given not infrequently, and such cases are to be classed with those following sudden frights. Peripheral irritation, as for instance, intestinal worms and nasal pharyngeal disorders, have been assigned as causes of chorea, but there is very little evidence for this, and the same remark applies to the alleged influence of hypermetropia and hypermetropic astigmatism and other forms of "eye-strain." The association of chorea with inflammatory affections of the joints, pericardium, and heart, has long been recognized, and it is customary to speak of the arthritis and cardiac inflammation as rheumatic; this point will be considered subsequently, but it may be said now that rheumatic fever does not appear to predispose, distinctly, to chorea. A recent attack of scarlet fever, more rarely of measles, diphtheria, typhoid fever, or septicæmia is sometimes assigned as the cause. On the other hand the onset of an acute exanthem during the course of an attack of chorea commonly suspends the movements. A great

deal of importance has been assigned to anæmia as a factor in the production of chorea, but on insufficient grounds; children frequently become anæmic during the attack. In most of the cases seen beyond the age of puberty the patient is found to be anæmic at the time of onset, but this certainly is not the case in the majority of young children attacked. Hysteria produces conditions resembling chorea, but does not play any direct part in the production of the disease itself.

[The observations of many American writers with regard to the frequency of anæmia in chorea do not coincide with those of the author. Rachford especially emphasizes the frequency and importance of this condition as an etiological factor in chorea and other neuroses in childhood. Though not the essential factor, it probably is one of the steps leading up to the explosion of the symptoms known as chorea. For, as a result of the anæmia, the nervous system, as well as all other parts of the organism, is in a condition of impoverishment, of partial starvation, and hence is unable to perform its functions properly. The cells of the nervous system in the child at best are in a condition of extremely unstable equilibrium on account of the rapid development and changes which the young organism is undergoing. In the anæmic state this instability is greatly increased; the nerve cells are more than ever sensitive and susceptible to external and reflex causes. For the same reason the higher centres have less inhibitory power than normal. In this way anæmia acts as an important predisposing factor in the causation of chorea.]

Pathology.—Although much has been written upon the pathology of chorea, nothing is as yet known with certainty. The occurrence of arthritis, pericarditis, and endocarditis indicates that chorea has a close nosological relation to rheumatism, but the evidence is by no means clear that chorea is merely a rheumatic manifestation. To quote the words of Osler.[1] "If, as some would have it, chorea is only one of the rheumatic states, we have to stretch beyond recognition our conception of the disease, now, in the absence of a knowledge of its etiology, necessarily characterized by its symptoms. Very probably the cause of chorea will be found to be a poison allied to, but not the same as, that of rheumatism." As to the nature of this poison nothing is known, though probabilities point strongly to its being a micro-organism capable of multiplying within the body; various pyogenic micro-organisms have been mentioned, and Pianese has isolated and cultivated a bacillus from the nervous system of a case of chorea, which when injected into animals caused death preceded by muscular twitching and convulsions. The evidence at present, however, is inconclusive. The frequency with which inflammatory affections of the joints occur in the course of various

[1] "On Chorea and Choreiform Affections" (London: H. K. Lewis, 1894).

acute infectious diseases should be borne in mind in this connection. There is much to be said for Sahli's theory that chorea belongs to the group of diseases of which septicæmia is the most typical member, but it would be a mistake to push the analogy too far, and Dr. Sturges thinks that "the heart symptoms of chorea seem best explained upon the hypothesis of some pathological kindred between it and rheumatism." The morbid anatomy of chorea does not throw much light on its pathology. The fatal cases, which are very rare, occur generally at or about the age of puberty; the frequency of death is eight times greater between the ages of 15 and 20, taking into consideration the number of cases, than under 10 years of age. The most constant lesion is endocarditis, which affects, in the vast majority of cases, the mitral valve, producing a row of small vegetations just within the margin of the auricular surface of the cusps. In a few cases death has been proved to have been due to malignant endocarditis, and there is some reason to believe that this is a somewhat more frequent cause of death after chorea, perhaps months after, than has been supposed hitherto. Next to the mitral the valve most often affected is the aortic, but it seldom suffers alone. At the same time endocarditis is not an essential part of chorea, since in some cases of death due to chorea in its most typical form, endocarditis has not been found *post-mortem*. Pericarditis is a rare complication; when it occurs it is generally associated with endocarditis. The most constant morbid conditions observed in the central nervous system are those indicative of hyperæmia—distension of the perivascular spaces, which contain many round cells, small areas of softening, minute emboli, and hæmorrhages.

The part of the nervous system primarily affected in chorea is unknown, but the marked psychical symptoms, the cessation of the movements during sleep, and the frequency with which they are either limited to one side or are greater on one side than the other point to the cortex. As Gowers has observed it is in the motor area of the cortex that movements are arranged, "and if they are disarranged the disorder proceeds from the brain, and we naturally refer it to a disordered action of the cells of the cortex." A cloudy swelling of the pyramidal cells has actually been described. The theory that the lesions of the central nervous system are due to "showers" of minute emboli, derived from the vegetations on the mitral valve must be mentioned, but the evidence in its favor is insufficient.

The condition of the *heart* in chorea is a point of much importance. Acceleration, unevenness of rhythm, and variability in force are almost constant phenomena; they are aggravated by any excitement, and may disappear after a little rest in the recumbent posture. In addition, in about a third of the cases a distinct murmur, systolic

in time, is heard, in most cases best or only at the apex. More rarely, a systolic murmur is heard at the base, generally in the pulmonary cartilage, but sometimes at the aortic, and along the left side of the sternum in the second, third, and fourth spaces. The murmur heard in the region last-mentioned is probably in most cases functional, that at the apex more rarely. The functional murmur is to be attributed either to functional insufficiency of the cardiac muscle ; to anæmia which, however, is not a common accompaniment of chorea, at least in the early stage ; or, perhaps, to the general toxæmia, which upon one theory is present in chorea. The systolic apex murmur is more often met with the younger the child, and the earlier the stage ; it may disappear before the movements cease. It is in some cases functional, but in others it is associated with endocarditis (beading of the mitral valve or marked endocarditis), but recent statistics [1] appear to prove that in more than half the cases of chorea some permanent damage of the mitral valve remains, though it may only become evident after some years. Cases of chorea distinctly complicated with arthritis are, however, those most likely afterwards to suffer from organic heart disease.[2] Whatever be the pathological nexus between chorea and rheumatism, the practical clinical point appears to be established that those cases which present distinct arthritis possess a less favorable prognosis as to permanent heart affections than others. Nephritis is an occasional complication.

In those cases in which the disease develops rapidly, and in which the movements are well-marked, the child is usually in an emotional state and appears to feel her condition acutely. She has a dull aspect, and is irritable and unable to concentrate attention. The movements cannot be controlled by the will, and are aggravated by observation or by exertion. Headache is severe in some cases. Fever is not observed in the majority of cases of chorea, but is present in some at the onset for a few days, but seldom reaches more than 100° or 101° F. Higher temperatures are usually due to endocarditis or some other complications. In very acute cases, how-

[1] Especially Osler's statistics from the Philadelphia Infirmary for Diseases of the Nervous System ; 140 cases were examined two or more years after the attack of chorea for which they had been under treatment in the infirmary. Of these 51 (36.4 per cent.) presented no signs of cardiac disturbance, 17 (12.1 per cent.) showed signs of cardiac disturbance believed to be functional, and 72 (51.4 per cent.) signs of organic heart disease. Of these 72 cases 25 had had acute arthritis (34.13 per cent.), so that there remained 47 cases which had suffered from chorea, but not from rheumatism, who yet presented signs of organic disease of the heart, that is to say about one-third of the total number of old cases of chorea examined.

[2] Donkin, "Diseases of Childhood" (London, 1893), examined 44 cases two years or more after they had been under treatment for chorea as in-patients ; of these 18 had had rheumatism at some time, and 13 presented persistent murmurs ; in 26 there was no history of rheumatism and only 5 had a murmur at the time of examination, and in three of these the murmur was not improbably hæmic.

ever, with delirium the temperature may rise to 105° F. In the majority of cases the movements affect all four limbs, and frequently the face and tongue also, but in not a few they are confined to the limbs and face on one side, or are much more marked on one side than on the other. In many of these cases of *hemichorea* paresis of the affected parts is marked and may be the first symptom, so that the patient is brought for treatment because it is noticed that one hand is weak, and that objects taken into it are dropped. Chorea, especially in girls about puberty, may be complicated by maniacal excitement, dependent apparently on an hysterical taint. The patient may be extremely violent, screaming and biting and scratching. The cases are, as a rule, in other respects mild, that is to say, the choreiform movements are not very severe. The term *chorea insaniens* is sometimes applied to these cases, as well as to those severe attacks in which there is fever accompanied by delirium.

The *course* and duration of chorea are very variable. Very mild cases may terminate in a few weeks; but not infrequently they drag on for months, temporary improvement being again and again followed by exacerbation, so that the child is never really free from the disorder for years. In a well-marked acute case of moderate severity, the symptoms generally begin to diminish in two or three weeks, and the whole attack is over in eight or nine weeks. Recurrence is, however, extremely common. Probably at least half the patients suffer one recurrence, many three or four, and some an even greater number.

In the **treatment** of chorea the most important element is rest for body and mind, and general experience confirms the statement of Osler that many cases which in the out-patient room seem very severe, become mild after a few days of rest in bed and seclusion from the anxious solicitude, or ill-timed severity of relatives. When the movements are very severe the patient must be protected from injury, and from falling out of bed, a not uncommon accident. The mattress should be soft, the sides of the bedstead covered with cushions, and furniture removed out of reach. Care must be taken to prevent bed-sores on the back, elbows, or legs. In these very severe cases it becomes imperative to give sedatives, potassium bromide or chloral, or a combination. Bastian's suggestion to keep the patient continually under the influence of chloral is valuable, and gives good results in cases which have resisted other forms of treatment; but the treatment is only called for in the worst cases in which the patient's life is threatened by want of rest. If the heart be feeble, as is often the case, alcohol may be required in pretty full doses. No remedy has any direct influence on the course of the disease nor on the severity of the movements, although arsenic has been praised as possessing power in both directions. To produce any decided effect,

however, on the movements it must be given in full doses, and has then been known to produce peripheral neuritis. In mild cases antipyrin is an excellent sedative, the dose being increased gradually until a distinct effect is noticeable. It is well, at the same time, to give some preparation of iron. Cod-liver oil should be given after the first severity of the initial disturbance has passed off. The child should be given a full and nutritious diet so long as fever is absent. Arthritis or endocarditis must be treated by sodium salicylate, but the effect of the drug is somewhat uncertain; as a rule, however, it allays pain. Swollen joints should be wrapped in cotton-wool. For the anæmia which remains after the movements have diminished or ceased, iron tonics and a diet containing a large proportion of fats is to be recommended. The child should not be allowed to return to school until the health has been completely re-established, and nutrition improved. Massage and regular gymnastic exercises, beginning with the simplest movements, are of value chiefly in the later stages when improvement has become arrested, and the movements threaten to become chronic. Benefit is also to be obtained from the galvanic current applied for ten minutes daily, using a large anode applied to the vertex, and the kathode in the hand.

CHAPTER XIX.

RICKETS.

Etiology—Pathology—Symptoms—Bony Deformities—Late Rickets—Complications —[Early Diagnosis]—Treatment.

Rickets (rachitis)[1] is a general disorder of nutrition, and its most characteristic symptoms are due, probably, to absorption of poisons produced in the gastro-intestinal canal by imperfect digestion. It manifests itself first, in most cases, during the second six months of life, about the time of the first dentition or a little earlier. In England, France, and Germany it is extremely common among the children of the working and poorer classes in towns, but is far from uncommon in the well-to-do class and in country districts.

The **etiology** of the disease is somewhat obscure. Improper feeding is a determining cause, and overcrowding, want of sun and air, and other unhygienic conditions, are contributory causes. There must also be a special predisposition, which may be congenital, perhaps hereditary, or acquired after syphilis, measles, and other acute infectious diseases. The initial error which sets going the processes which result in rickets is the use of a diet deficient in some essential constituent. Though a defective diet may not by itself be sufficient to produce rickets, it is comparatively rare to meet with cases in which there is no history of bad feeding. Breast-fed children commonly escape, and when they do suffer they are generally the offspring of mothers exhausted by frequent pregnancies and prolonged lactation. Harassing work under bad hygienic conditions and insufficient food are other unfavorable conditions of the mother's life which favor rickets in her child, and to their operation, and perhaps also to the influence of heredity, are to be attributed the occasional cases of rickets in first-born children suckled by the mother. It is common to find that a child suffering from well-marked rickets has been weaned early from the breast (within the first three or four months of life), and has been brought up on condensed milk, or very much diluted "fresh" cow's milk, thickened with some starchy food, "perhaps prepared" by conversion of part of the starch into dextrin. [Observation of many cases of rickets in infants, both at the breast

[1] A word coined, according to Skeat, about 1650, with a punning allusion to Gk. *ῥάχις*, the spine. *Rickety* is an English word signifying tottering, from Middle English *wrikken*, to twist.

and on the bottle, has convinced the reviser that in a large majority of the cases deficiency of fat is the most important etiological factor, and that rachitis in its earliest stages is in reality "fat-starvation." Analysis of the breast-milk in the breast-fed cases invariably shows a low percentage of fat, and the various "foods" are notoriously deficient in this element. It is not an uncommon thing to see rickets developing in children at the breast, food from this source also being often entirely insufficient for the purposes of nutrition; hence we see in such infants some form of mal-nutrition developing and most commonly rickets.]

Pathology.—The disease occurs at a period of life when the bone-forming tissues are most active, and the stress of defective nutrition falls particularly on them, causing excessive and irregular growth, accompanied by deficient calcification. In health these tissues are found in three situations in a long bone: (*a*) immediately beneath the epiphysial cartilage—the chondroid layer; (*b*) immediately beneath the periosteum; and (*c*) the medulla. In a rickety long bone the chondroid layer is unnaturally thick and vascular, and instead of abutting directly upon hard bone, ends by an irregular edge in spongoid tissue which consists of imperfectly formed trabeculæ, containing little lime, and loosely arranged so as to leave large alveoli containing a soft red marrow. The sort of cavernous structure thus produced has led to the use of the term spongoid. The subperiosteal bone-forming tissue is also increased in bulk and vascularity, so as to form a soft layer, thickest in the middle of the shaft. It is so vascular, as well as soft, as to resemble a subperiosteal hæmorrhage, and to render the periosteum easily detachable. This is the "osteoid tissue" of Virchow; eventually it becomes very hard bone. There is also increased vascularity and overgrowth of the medullary tissue of the centre of the bone, and this leads to absorption of good bone already formed. Later this loose-structured medullary tissue is transformed into fibrous tissue, and finally into very hard, ivory-like bone. When this change is extensive and occurs early, the functional activity of the chondroid layer is destroyed, the epiphysis becomes firmly attached to the shaft, and the bone can no longer increase in length. This is one part of the mechanism by which rickety dwarfs are produced. In cases which run a more favorable course the spongoid tissue is gradually replaced by true ossifying tissue. The chemical changes which attend these anatomical lesions are, in the main, a diminution of the lime salts (to as little as one-third of the normal), and an increase in the amount of water.

The bony deformities of rickets depend upon two causes: (1) the actual increase in bulk; and (2) the softening of the bones, which allows them to become distorted by muscular action or by the weight of the body.

Symptoms.—Rickets varies very much in intensity. The *invasion* may be rapid and attended by fever, gastro-intestinal disturbance (thirst, lost of appetite, diarrhœa, abdominal distension), rapid enlargement of the epiphyses of the long bones, bossing of the cranial bones, and great tenderness of the whole osseous system. The skin is warm and perspires readily. The sweating is particularly copious at night, or at any time when the child sleeps. It is especially free from the head, so that the pillow is drenched. The child is very restless when awake, unable to find an attitude in which it can be comfortable. Sleep is not sound, the eyes are half shut, and there

FIG. 18. FIG. 19.

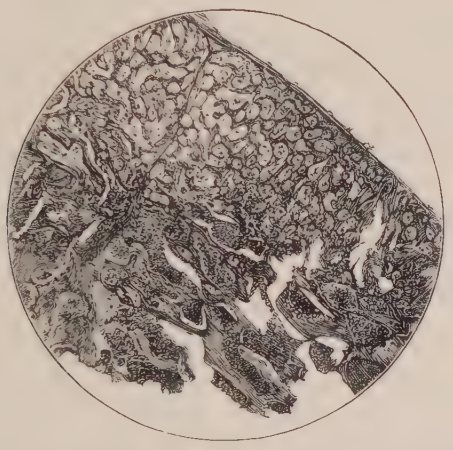
A boy aged 15 months, fairly well nourished, presenting rickety conditions of moderate degree. Fig. 18 shows the attitude in sitting, but one hand has been raised to exhibit the swelling at the wrist. Fig. 19 shows also the backward excurvation of the spine.

are constant restless movements. These symptoms are to be traced to tenderness due to the active changes occurring in the bones. The tenderness and the heat of the skin lead the child to kick off the clothes. The temperature is higher than normal in the evening. Frequently the onset of rickets is very insidious, more often than not the earlier symptoms pass unobserved, and the child is first brought for the treatment of bronchitis or some other complication, with the bony changes already developed.

The *developed* disease is always chronic, and the softness of the bones, the sweating, and the gastro-intestinal derangement may per-

sist for months or years, often till the end of the long-delayed first dentition. By the action of various mechanical agencies numerous deformities are produced. In the *skull* the disease itself produces a thickening and softening of the bones. This change is general, but the thickening is most marked at the edges of the anterior fontanelle, and at the frontal and parietal eminences. In other cases there is a central frontal boss which gives to the skull a peculiar elongated carinate appearance. When the bossing of the eminences is well marked the skull assumes a peculiar and characteristic shape to which the term natiform has been applied. Associated with this bossing, though seldom when it reaches an extreme degree, areas of thin bone may be found. The term *cranio-tabes* is applied to this

FIG. 20.



Section of a part of a cranial boss, from a case in which cranio-tabes was also present. (Photomicrograph by MR. F. FOWKE.)

condition. It is an early sign, and there may be at first, at least, no obvious thickening of the edges of the anterior fontanelle nor bossing of the skull. It probably occurs more often in infants who have suffered from syphilis than in others. Cranio-tabes is met with usually towards the back of the head, but it may involve almost any part of the cranial vault. When the change is well marked the bone yields and as it were crackles under the finger like parchment, often over large areas. In some cases, most often those in which there is a good deal of thickening in other parts, the bone in the thin areas is finally completely absorbed. The pressure of the head on the pillow may produce very considerable distortion of the skull, a flattening in the antero-posterior direction, and a broadening from side to side. When well developed, cranio-tabes permits considerable pressure to

be exerted, which interferes with the intracranial circulation. To this cause some are disposed to attribute the special liability of rickety children to laryngismus stridulus, to eclampsia, and to tetany, and they point to the visible distension of the superficial veins at the base of the skull and of the jugular vein in support of the view. The nervous symptoms mentioned, as well as the venous distension, may also be observed in children who do not suffer from cranio-tabes, but in whom the skull appears to be everywhere much thickened. It is unlikely that in such cases any pressure can be exerted on the cranial contents, and it has even been maintained that the brain of a rickety child grows with undue rapidity because the cranium, owing to the softness of the bones, can be easily distended.

The first *dentition* is delayed. The first tooth is cut late, the intervals between the appearance of the other teeth are prolonged, and the natural order of their eruption is disturbed. The teeth are not well formed. The cutting border of the incisors is rounded and blunt, though this appearance is apt to be modified by the early occurrence of "erosions." The enamel is deposited irregularly, and stops short suddenly at the neck. The erosions are of two kinds : (1) pits due apparently to defects in the enamel ; (2) transverse striations, which may be so deep and numerous as to reduce greatly the thickness of the tooth. These striations are probably produced by stomatitis occurring at about the time when the tooth is being cut. In many cases the teeth assume quickly a yellowish color and decay rapidly. They may even be carious when cut, and it is not uncommon to see all the incisors reduced to mere brown stumps, which, however, do not seem to be tender or painful. The form of the jaws becomes altered. The lower jaw becomes angular, and square in front, compressed at the sides, and the alveolar border is bent inwards. The alveolar border of the upper jaw is bent forward and the palatine arch is high. The two last mentioned deformities have been attributed to the pressure of the tongue in sucking. Enlargement of the bones of the face is not conspicuous, and the contrast between the large, flat, bossed cranium, and the small emaciated face is in some cases striking.

The deformities of the bones which are of greatest importance as they affect the prospect of life are those of the *thorax* (Fig. 18). The simplest form consists merely of a rounded thickening at the costo-chondral junction. This row of knobs on either side of the chest is the " rickety chaplet," one of the earliest and most constant signs of rickets. [We cannot agree with this opinion. See below under " Diagnosis."] It corresponds to the enlargement of the epiphysial ends of the long bones. It is not in itself of much importance, but it indicates the commencement of changes in the ribs which will render possible the production of deformities diminishing greatly the

capacity of the chest. Kyphosis (Fig. 19) is a deformity which commonly develops early; eventually the spine may form one continuous backward curve from the neck to the sacrum—"cat's back." The angles of the ribs become unnaturally acute, and the horizontal section of the thorax tends to the form of an equilateral triangle, with the apex at the sternum. The thorax may remain symmetrical, but more often there is some prominence on the left side in front corresponding to the precordial region, and at the same time some lateral curvature of the spine with the convexity towards the right. In association there is some flattening of the angles of the ribs on the left side, and increased acuteness and prominence of the angles on the right. The cause of this lateral distortion is not very clear, though it would seem sometimes that the asymmetry originates because the heart offers greater resistance to compression than the lungs. Lateral curvature with convexity to the right is, however, far from invariable, and if the child is carried habitually on the left arm of the mother the scoliosis may be in the opposite direction. This deformity is produced by the child's body being bent round the mother's chest. Under the influence of any disorder which causes difficulty in respiration further and more marked deformities may be produced. One of these is the formation of a deep vertical groove due to a bending-in of the ribs a little behind the costo-chondral junction. This throws forward the rib-cartilages and the sternum. The sternum also may be bent forward at the junctions between its several parts. In this way a form of pigeon chest is produced. A second deformity is the formation of a horizontal groove corresponding approximately to the insertion of the diaphragm. In forcible inspiration the softened ribs tend to bend inwards, and, with imperfect expansion or collapse of the lower portions of the lungs, there is nothing to prevent this tendency until the level of the abdominal viscera, the solid mass of the liver and the stomach and colon often distended, is reached. The lower part of the thorax is held out by the abdominal viscera, the upper part by the upper lobes of the lungs, which are well expanded or perhaps emphysematous, and a groove forms in the intermediate region by the falling in of the ribs. The combination of these deformites produces the most characteristic rickety aspect—the small chest, prominent in front, with the rickety chaplet, horizontal groove, and distended, almost globular, abdomen. The pelvis is liable to undergo considerable deformity. It becomes shallower, and the iliac crests are nearer together than natural, while the antero-superior iliac spines are bent outward. The cavity of the pelvis may be much narrowed by inward projections corresponding to the hip-joints, and sometimes also by a pressing forward of the sacrum. The lower aperture of the pelvis is not narrowed. In the female these deformities may be the cause of difficult labor.

The deformities in the *long bones* over and above the enlargement of the epiphyses and of the shaft, are produced by external mechanical agencies, and their character depends upon the habits of the child. In an infant, which for the greater part of its life lies on its back in a cradle, they will be little marked. In an infant which is carried habitually by its mother, some bending of the long bones, those of the lower limbs especially, but also of the arms, will be produced. The exact character of the deformity depends on the attitude in

FIG. 21. FIG. 22.

Rickety Deformities. Fig. 21 shows especially lordosis and forward bending of the femurs. Fig. 22, from the same patient, genu valgum and the small, grooved chest. (From photographs by DR. PERCY C. PHILLIPS.)

which the infant is carried and suckled. In a child which can sit up great deformities may be produced. When well marked they are very characteristic. The child sits with its legs crossed in the attitude assumed by tailors—that is to say, with the knees flexed and one leg crossing the other at, or just above the ankle (Figs. 18–19). The hands rest usually with their palmar surfaces on the bed beside the hip-joints. The heels are drawn up close to the perineum, and the weight of the child is borne mainly on the nates and the hands. In consequence, excurvations of the bones of the upper limb, but especially of those of the forearm are produced, so that the whole

limb is bowed outwards. In the lower limbs the main deformity is in the bones of the leg, and is produced at the point where the legs cross each other. If, as is often the case, the child sits habitually with the same leg uppermost, the tibia of this leg is bowed inwards (upwards as the child sits) and other leg outwards (downwards as the child sits). The deformity in the femur is less, and consists in a bowing forwards; children who have acquired this attitude seldom learn to crawl; their earliest mode of progression is to push themselves

FIG. 23.

Skiagram showing the curving of the bones in a case of genu valgum. (MR. C. A. MORTON'S case.)

along the floor in a sitting posture throwing the weight of the body mainly on the arms, and progressing by working the nates forward, first on one side and then on the other. When the child begins to walk early it often becomes bow-legged. This habit tends to maintain and aggravate the deformity of the upper limbs. After the age of infancy the rickety deformities of the lower limbs may be attended by lordosis (Fig. 21), and in many cases knock-knee is produced (Figs. 21, 22, and 23).

In rare cases rickety deformities begin to make their appearance at the age of twelve years or later. The causes of the condition appear to be similar to those which produce rickets at an earlier age, and the pathological process is identical. Such cases are therefore justly denominated *late rickets*.

Among cases of rickets two clinical types are generally recognized —small and large. In *small rickets* the child is small and thin and light for its age, and often precocious. The enlargement of the bones is confined commonly to the epiphyses and is not extreme. In *large rickets* the child is big and fat, and dull rather than precocious. The cranial and other bony changes are well marked, and the copious perspirations are a source of much discomfort. Small rickets is met especially among the neglected children of the poorest class; their food is often defective, not only in quality but in quantity, and they suffer from chronic diarrhœa. The child with large rickets is commonly well cared for, and fed copiously on condensed milk and prepared food. It is seldom free from some bronchial catarrh, and suffers from frequent attacks of bronchitis.

Complications.—Gastro-intestinal catarrh is a common accompaniment of all stages of rickets. Dyspepsia and diarrhœa are so frequent at the onset of the disorder that they have been regarded as the determining causes. At a later stage catarrh of the large intestine is very common, and the stools are frequent, consist largely of mucus, and are often very foul-smelling. Lienteric diarrhœa is not uncommon, and in other cases the child suffers from alternating diarrhœa and constipation. Rickety children are very liable to suffer from diarrhœa in hot weather, and slight errors of diet will in them determine an attack. They are especially liable to bronchitis and broncho-pneumonia. These diseases are particularly dangerous in them, owing to the diminution in the capacity of the chest, which, if it does not already exist, is quickly produced. Owing to the readiness with which the chest walls yield, bronchitis is peculiarly liable to be complicated by collapse, which paves the way for broncho-pneumonia. For the same reason the pulmonary circulation quickly becomes embarrassed, and dilatation of the right side of the heart contributes to bring about a fatal termination.

[**Diagnosis.**—Too much importance cannot be attached to an early diagnosis of rachitis, for it is in its earliest stages that treatment is most successful. It is a preventable disease, provided the child be born healthy. If the diet be carefully regulated and the infant receive the proper amount of fat and albumenoids, either in breast-milk or modified cow's milk, rickets will not develop. If, however, the child fail to receive a proper amount of these elements he will soon show signs of disturbance. If rickets be developing, head-sweating, restless sleep and constipation will soon be noted. The

occurrence of these three symptoms in a nursling should at once put us on our guard and lead to an investigation of the infant's food, and wherever possible a chemical analysis either of the breast-milk or of the bottle-food. They are the earliest signs in the development of rachitis, occur long before bone changes are manifest clinically and should serve as a danger-signal, foretelling serious disturbance in the child's nutrition. If analysis of the food show a diet deficient in either fat or proteids, we may feel sure that sooner or later the characteristic bone-changes will follow. The diagnosis then is of course an easy matter, but by that time much mischief has been done which could have been prevented. Careful inquiry into the details of the infant's life will too often bring out the above tripod of symptoms, and it is at that stage that our treatment should be instituted. The physician is rarely consulted for these conditions, but he will find them on investigation when called for some intercurrent disease. Especially will he find them in the daily routine of large city clinics among the half-starved nurslings of equally half-starved mothers. The occurrence of rickets in infants fed on one of the so-called "baby-foods" is too common to need more than a passing mention.]

The **treatment** of rickets consists in the main of the correction of these errors of hygiene and diet which are believed to be its chief causes. The child should live in well-ventilated rooms and should have *fresh air* daily; if it be suitably clothed in woollen garments, there are few days in the year even in a British climate when it may not be out for several hours. While active softening of bones is in progress it should be carried as little as possible in the arms, and should go out in a cradle-perambulator. The *diet* must be carefully inquired into, and any errors corrected. Milk is the best food, but one or two meals a day of well-cooked fine oat, or other whole meal, may be given after the age of one year, or even a little earlier. If the patient is an infant at the breast it should not be weaned until after the age of twelve months, unless the mother's health has become much deteriorated; and even so, it is often better to endeavor to improve her nutrition, and to supplement the breast-milk than to wean the infant. At a more advanced age the child should have a nutritious diet containing a large proportion of fats and of proteids. Starchy foods should not form the staple article of diet. Fresh fruit is well taken if there be no great gastro-intestinal irritation, and when the patient is anæmic its use forms one of the most important parts of treatment. The child should have a *bath* daily; this must be warm for infants and for fragile children at all times of the year—but cold effusions to the limbs should be used if they do not cause the patient too much distress. Salt baths are also of use, owing to their stimulating effect on the skin. Change of air, especially to the *seaside*, is

very beneficial, and then warm sea baths should be prescribed. *Massage* of the limbs is useful, and should be used except when the tenderness is too acute. Diarrhœa, bronchitis, and other complications must be treated by the remedies appropriate to the several forms of these disorders, as indicated elsewhere. Atropine (gr. $\frac{1}{100}$) may be given in the evening to check sweating. It is essential to get the digestive system into good order before any permanent benefit can be expected from other remedial measures. No *drug* has any specific action on the course of the disease. Phosphorus, for which this power has been claimed, is of value only during what may be called the acute stage, when tenderness is extreme and copious sweats are undermining the strength and exposing the child to the risk of catching cold while out of doors. It may be given in cod-liver oil, or, if this cannot be tolerated by the stomach, in solution. The dose should be from $\frac{1}{120}$ to $\frac{1}{60}$ grain. [One of the following preparations may be used:

Tincture Phosphori Composita (B. P. C.).

Phosphorus,	gr. ij
Chloroform,	ʒv

Warm gently in a stoppered bottle till dissolved, and add the solution to ethylic alcohol ʒxv. Shake and keep in the dark (1 in 600).

Elixir Phosphori (B. P. C.).

Tr. Phosphori Co.,	ʒj
Glycerine,	ʒiv

To be prepared freshly: contains gr. 1/50 in ʒj. Dose for an infant ♏x–xx.

Oleum Morrhuæ Phosphoratum.

Ol. Phosphorati (B. P.),	ʒij ♏xl
Ol. Morrhuæ,	Oj

Contains 1/100 in ʒj, which is the dose. Appendix.]

The greatest benefit is derived from the continuous administration of cod-liver oil, which may almost be claimed to be a specific. It should be given at first in small doses, which should be increased as tolerance is established. An infant of one year, if its digestion be in good order, can usually take a drachm twice or even thrice a day. It should be given alone or with malt extract, or if this fail, in emulsion, guarded, if there be tendency to diarrhœa, by half a drop of tincture of opium. If after a fair trial it appears that cod-liver oil cannot, at the time, be borne, it may be replaced by a syrup of the phosphate of iron, or by the ammonio-citrate of iron. Deformities of the long bones must be treated by suitable splints and by massage so long as the bones are still soft. At a later stage, when osseous sclerosis has occurred, the question of osteotomy or osteoclasis will arise; but when treatment, hygienic and local, can be carried out early with sufficient perseverance and skill, very remarkable improvement may be obtained without surgical interference.

CHAPTER XX.

SCURVY.

Age—Etiology—Morbid Anatomy—Symptoms—Course—Treatment.

No age is free from the liability to scurvy, but in infancy the disorder presents certain special characters, owing, apparently, to the great physiological activity of the growing parts of the bones at that age, and the peculiar liability to rickets.

Age.—It is rare for a child to suffer from scurvy before the age of nine months, and the period during which the disorder is most often seen is between that age and about eighteen months.

Etiology.[1]—The age at which infantile scurvy has been observed most often, covers the period during which a child weaned early from the breast is most likely to be kept on a rigid diet. The disease is certainly more common among the children of the well-to-do classes. This fact is to be attributed to the prevailing habit of feeding infants entirely on proprietary foods made of starch, and on sterilized or condensed milk. The children of the poor may have similar preparations in their bottles, but they begin at a very early age to pick up a certain amount of food at the general table, whereas with the strict discipline of a well-dragooned nursery this natural habit is suppressed. Condensed milk, and the various kinds of commercial milk preparations sufficiently well sterilized to keep for months, as is the case with some humanized milk, are also causes of scurvy if used to the exclusion of fresh milk. Even fresh milk, if too much diluted, may not prevent the onset of scurvy, and the same may be said of scalded milk, and perhaps of milk sterilized for short periods, though not of pasteurized milk. Want of fresh air and sunlight may be contributory causes, but syphilis, rickets, and other constitutional disorders which produce marasmus, do not produce scurvy unless fresh food is deficient. The actual onset of the attack appears to be determined in some cases by an injury, in others by a sudden increase in the dilution of the food given.

Morbid Anatomy.—The characteristic lesion is subperiosteal hæmorrhage, but this may be absent if there be no rickets. The perios-

[1] Barlow's views as to the Etiology and Pathology of Infantile Scurvy, though not universally accepted, appear to me to be correct, and have been followed here. See his Bradshaw Lecture (*Brit. Med. Jour.*, 1894, vol. ii., p. 1029).

teum is intensely vascular, and the amount of effused blood between it and the shaft of the bone may be very considerable. Hæmorrhage takes place also into the cavity of the shaft, causing destruction of trabeculæ and leading in time to absorption of the compact tissue. Blood is effused also into and between the muscles near the bone, and serous effusion is found in their more superficial parts. Proptosis is due to extravasation between the periosteum and the roof of the orbit. The swellings over the cranial bone are due to extravasations under the scalp, generally in relation with portions of bone previously rendered unduly vascular by rickety changes. Hæmatoma of the dura mater also may be produced. Hæmorrhage, which may be extensive, may also occur into the lungs, and minute hæmorrhages may be found in the intestinal mucous membrane, mesenteric glands, and the pyramids of the kidney. Occasionally small hæmorrhages take place into the joints. In a case which has reached a more advanced stage, and in which the process is receding, a layer of bony tissue will be found on the under detached surface of the periosteum, and the muscles will be much wasted.

Symptoms.—The previous history of the child may have been quite uneventful, or there may have been much difficulty in finding a food which it could digest. In any case it will have been hand-fed. The first symptom noticed is that the infant becomes difficult to handle ; it cries whenever it is touched, but is fairly quiet when left lying down. It keeps the lower limbs drawn up, and it is evident that they are tender. A little later some swelling—first of one lower limb, thigh or leg, then of the other—can be detected. There is no local heat, redness, or pitting, and the swelling, which is not well defined, appears to be in relation with the shafts of the bones. Later, the swelling in these situations becomes greater, there may be a little œdema, and the skin may have a purplish tint, but this is seldom very pronounced. The attitude in which the child lies is changed, the legs are everted and motionless, so that the infant is thought to be paralyzed, an opinion apparently confirmed by the weakness of the back. Some swelling of the upper limbs, above the wrists, and near the epiphyses of the humerus, may now be perceived, and also perhaps some fulness over the scapulæ. The lesions, though not exactly symmetrical, are yet commonly present on both sides. The joints are not affected, but at an advanced stage of the malady crepitus may be obtained at about the junction of the epiphysis and diaphysis of the femur (upper and lower) and tibia (upper) or more rarely the humerus. Occasionally, fracture occurs in the middle of the shaft of the femur. A peculiar deformity of the chest may appear ; the whole front of the thorax, including the sternum, costal cartilages, and the anterior parts of the ribs, sinks in as though the ribs had been fractured in a row just beyond the costo-chondroid junctions,

which indeed is the case. In a few instances areas of thickening may be made out over the cranial, and sometimes even the facial bones. The areas are tender, but there is no local heat and seldom any change of color. Proptosis first of one and then of both eyeballs, with puffiness and some staining of the lids, may develop somewhat suddenly, with perhaps some ecchymosis of the ocular conjunctiva. The state of the gums depends upon the stage of dentition which has been reached. If no teeth have been cut, the gums are normal, or present only some purple stains where the teeth are about to appear. If a few teeth have been cut, they will be surrounded by narrow fleshy ridges; if many, the sponginess of the gums may be so great that fleshy masses protrude from the mouth. Such spongy gums bleed easily, give rise to much fœtor, and interfere with feeding. Epistaxis may occur, and occasionally blood has been passed from the bowel, but digestive disturbance is not a marked or a necessary symptom, though loss of appetite is the rule. The urine often contains a trace of albumen, sometimes blood. The heart and lungs show no signs of disorder. The spleen may be a little enlarged. The most marked general symptom is anæmia, which is proportional to the degree in which the bones are involved. The infant becomes very pale, and after a time the skin assumes an earthy tint, and petechiæ, or larger ecchymoses appear. There is also extreme prostration, though there may be little or no emaciation. There is no regular pyrexia, but as each fresh limb is attacked the temperature may rise for a day or two to 101° or 102°F.

Course.—If untreated the cachexia becomes very profound, and the infant commonly succumbs to some intercurrent affection—bronchitis, pleuro-pneumonia, diarrhœa, or a specific fever. If these dangers be escaped, improvement begins in two, three, or four months. The swelling of the limbs, and with it the tenderness, diminishes, leaving a firm swelling around those parts of the bones which were most affected. This is the more evident owing to wasting of the muscles. After a time fractures, if they have occurred, unite without much deformity, except those which involve the middle of the shaft, where much callus may be thrown out. Anæmia and asthenia disappear gradually. On the other hand, under proper treatment recovery is very rapid.

The **symptoms** detailed are those of well-marked cases, but it is probable that mild degrees of scurvy are responsible for cases in which with moderate signs of rickets there is excessive tenderness, and some anæmia, symptoms which are quickly relieved by giving fresh food. Hæmaturia may be the solitary sign of scurvy, and possibly also proptosis.

The **treatment** is simple and satisfactory. The infant must be put upon a diet of fresh food suitable to its age. Fresh whole milk is

the best. It should be given undiluted. To it, or to milk diluted not more than by the addition of one part of water to three of milk, may be added a little sieved potato. A tablespoonful of orange or grape juice may be given in water during each day, and to children a little older about the same quantity of fresh meat-juice, or gravy.

[**Raw Meat-Juice.**—Mince fine one-quarter pound best rump-steak, free from fat and gristle, add two tablespoonfuls of water, stir, and set aside for one hour. The juice is expressed through muslin by twisting. From 2 to 3 fl. oz. may be given in twenty-four hours. It may be given in milk, the taste of which it does not much modify. (Cheadle.)

Raw Meat Pulp.—Take 2 oz. of best rump-steak, scrape fine with a knife on a cook's board, removing all gristle and fat. If not quite pulped, pound in a mortar. May be taken alone, mixed with a little finely minced parsley (about half a teaspoonful), or spread between thin slices of bread. At one year, this quantity may be given during the day. Appendix.]

The child should be kept at rest in the horizontal position, which not only prevents him from suffering the pain which handling causes, but obviates the danger to heart failure which is liable to occur in extreme anæmia and debility. All the symptoms diminish rapidly under this diet, and in a short time it may be necessary to reduce the amount of fresh undiluted cow's milk and vegetable which is at first greedily taken and assimilated with ease. Local conditions require treatment only in extreme cases; thus it may be necessary to apply splints for fracture, and to use antiseptic and astringent applications for bleeding from the gums. Subcutaneous and periosteal hæmorrhages should be treated by gentle pressure maintained by pads of cotton-wool.

Prevention.—Scurvy seldom comes on before the ninth month. It is, therefore, about the eighth month that the diet of a hand-fed infant should be considered with a view to its prevention. If fresh milk can be obtained from a reliable source, and can be digested whole or very little diluted, it will be sufficient to continue its use. If, as is commonly the case in towns and during hot weather, the milk must be boiled, it may be thickened with freshly sieved potato. If this is not well taken, as sometimes happens when there is gastro-intestinal catarrh, fresh meat-juice or gravy, about a tablespoonful of either should be given daily. Frequently infants of a year old, or even less, will take fresh ripe fruit with pleasure, and without discomfort. Indeed, it is curious to see how many young children will suck with satisfaction at an orange or, even, a lemon, which is so sour that to the adult palate it is extremely distasteful.

CHAPTER XXI.

ANÆMIA AND LEUCHÆMIA—HÆMOPHILIA.

[Normal Blood in Early Life]—Secondary Anæmia—Primary Anæmia—Chlorosis—Progressive Pernicious Anæmia—Splenic Anæmia—Leuchæmia—Hodgkin's Disease—Hæmophilia.

Both the white and red blood-corpuscles and the amount of hæmoglobin are stated to be above the average at birth. There are also a few nucleated red corpuscles, but immediately after birth there is a diminution in all these constituents.

[This diminution, however, is not sufficient to bring the number to the average of adults, and, as we should expect from the generally undeveloped state of the infant and child, we find in the blood of these periods considerable variations from the normal as found in adults. The number of red corpuscles is generally somewhat higher, averaging a little over 5,000,000 per cubic millimetre. During the first few weeks of life, we see variations in the size and shape of the corpuscles and also see, more commonly, nucleated reds. The difference, however, is more marked when we come to an estimation of the various forms of leucocytes. The following table illustrates averages as given by Cabot, Gundobin, Morse, Rotch, and others:

	Infants.	Adults.
Small Mononuclear } young cells.	50–70 per cent.	24–30 per cent.
Large " " } young cells.	6–14 "	3– 6 "
Neutrophiles (adult cells)	18–40 "	60–75 "
Eosinophiles (old cells)	½–10 "	1– 2 "

In other words, we see an increase in the "young" and a diminution in the "adult" cells. These proportions it is important to bear in mind in making a differential count of the white cells. This count may be made as follows: Cover-glasses are cleaned with soap and water or with alcohol, the ear is punctured and a cover-glass touched to the drop of blood. Upon the cover, another is placed and the blood thus spreads out between them. They are then drawn carefully apart, heated over the flame of an alcohol lamp for thirty to sixty seconds and covered for the same length of time with the Ehrlich tricolor mixture, prepared as follows:

Ehrlich—Biondi Powder,	gr. xv
Alcohol (absolute),	1 c.c.
Distilled water,	6 c.c.
	(Cabot.)

Wash with water, dry and mount in Canada balsam, examine with the oil-immersion lens.

For more delicate work, it is advisable to heat the specimens for an hour at a temperature of 110°–115° C. and then to stain for three or five minutes, but for ordinary clinical work, the shorter method is sufficient.

The actual counting is simple and consists merely in checking off the different cells and putting them under their respective headings. Cabot states that one thousand cells should be counted.

Plate II., from Park's Surgery, shows the different types of leucocytes, varieties of red corpuscles, and the appearance of the blood in various pathological conditions.]

Very numerous investigations have been made upon changes in the blood produced by disease. The changes in the cellular constituents include diminution in the number of red cells, and in the amount of hæmoglobin, an increase in the number of leucocytes, and the presence of certain abnormal white cells. To all these conditions the term anæmia is commonly applied, though for that last mentioned the term leuchæmia is more appropriate.

[The changes are exaggerated and more marked in the blood of infants and children than in that of adults. The whole of the young

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DESCRIPTION OF PLATE II.

Blood.

Fig. I. Types of Leucocytes.—*a.* Polymorphonuclear Neutrophile. *b.* Polymorphonuclear Eosinophile. *c.* Myelocyte (Neutrophilic). *d.* Eosinophilic Myelocyte. *e.* Large Lymphocyte (large Mononuclear). *f.* Small Lymphocyte (small Mononuclear).

Fig. II. Normal Blood.—Field contains one neutrophile. Reds are normal.

Fig. III. Anæmia, Post-operative (secondary).—The reds are fewer than normal, and are deficient in hæmoglobin and somewhat irregular in form. One normoblast is seen in the field, and two neutrophiles and one small lymphocyte, showing a marked post-hæmorrhagic anæmia, with leucocytosis.

Fig. IV. Leucocytosis, Inflammatory.—The reds are normal. A marked leucocytosis is shown, with five neutrophiles and one small lymphocyte. This illustration may also serve the purpose of showing the leucocytosis of malignant tumor, except that in this disease (malignant) the reds show a well-marked secondary anæmia.

Fig. V. Trichinosis.—A marked leucocytosis is shown, consisting of an eosinophilia.

Fig. VI. Lymphatic Leukæmia.—Slight anæmia. A large relative and absolute increase of the lymphocytes (chiefly the small lymphocytes) is shown.

Fig. VII. Spleno-myelogenous Leukæmia.—The reds show a secondary anæmia. Two normoblasts are shown. The leucocytosis is massive. Twenty leucocytes are shown, consisting of nine neutrophiles, seven myelocytes, two small lymphocytes, one eosinophile (polymorphonuclear) and one eosinophilic myelocyte. Note the polymorphous condition of the leucocytes, *i. e.*, their variations from the typical in size and form.

Fig. VIII. Varieties of Red Corpuscles.—*a.* Normal Red Corpuscle (normocyte). *b, c.* Anæmic Red Corpuscles. *d-g.* Poikilocytes. *h.* Microcyte. *i.* Megalocyte. *j-n.* Nucleated Red Corpuscles. *j, k.* Normoblasts. *l.* Microblast. *m, n.* Megaloblasts. (Prepared by Dr. I. P. Lyon.)

Fig I

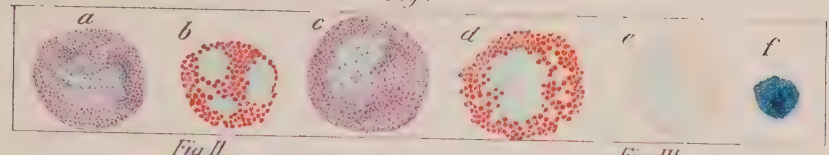


Fig II.

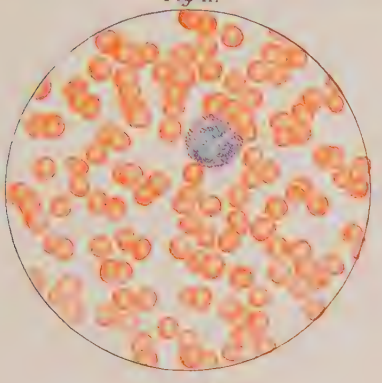


Fig III

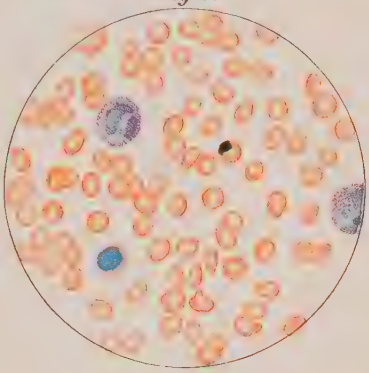


Fig IV.

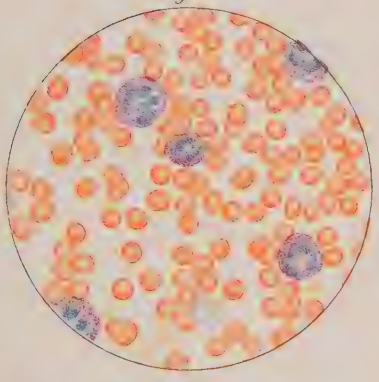


Fig.V.

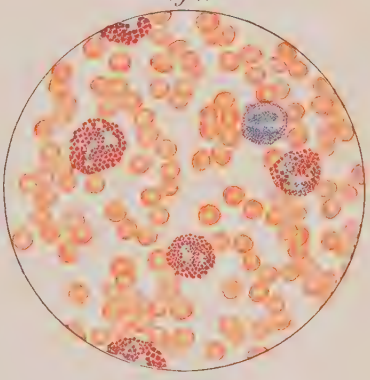


Fig VI.

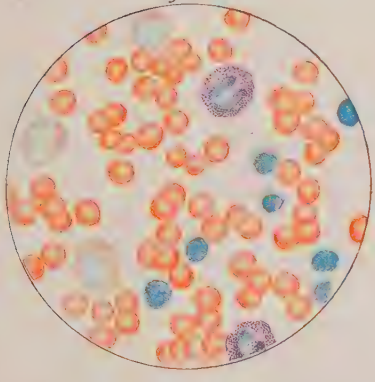


Fig.VII.

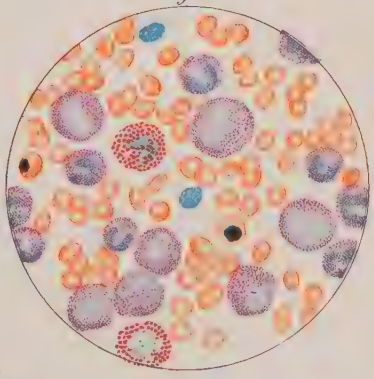
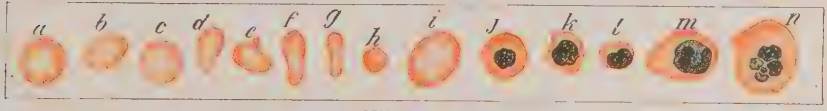


Fig.VIII



DRAWN BY J N Z CHASE

organism is more sensitive to, and more seriously disturbed by, morbid processes than is the adult, and the blood but shares in this general peculiarity. As in all the processes of these periods, both physiological and pathological, irregularity and instability are marked; hence slight causes produce greater disturbances. The blood, therefore, is easily affected, shows changes quickly, and the relative proportion of the whites is disturbed. There is a general tendency to revert to the fœtal type of blood. Leucocytosis is more easily produced and to a greater degree, and is generally a lymphocytosis, the infantile type. Splenic enlargement is common in all the anæmias of infancy.]

Anæmia may be primary, due to a disorder of the blood itself, or secondary, due to derangement of other organs of the body. The tendency of recent observations is to reduce the number of forms of anæmia which can properly be regarded as primary.

Secondary anæmia may be due to a variety of causes and is a common complication of a large number of diseases. It may be due to (1) *inanition*, in which all the elements of the blood are reduced; (2) to *hæmorrhage;* here the number of red corpuscles is at first reduced and those present vary in size and some are nucleated (see Fig. 3, Plate II.); the hæmoglobin is reduced in greater proportion than the corpuscles. The leucocytes are increased in number, owing mainly to the presence of a larger proportion of polynuclear cells; (3) *pyrexia*, from whatever cause, produces anæmia, owing partly to destruction of cells and partly to deficient production. It may or may not be accompanied by an increase in the number of white cells, which is observed especially in pneumonia, but also in pericarditis and endocarditis, in pleurisy, in pyogenic diseases including purulent meningitis, septicæmia and osteo-myelitis, in acute rheumatism, erysipelas, diphtheria, scarlet fever, and small-pox. In tuberculous diseases, including tuberculous meningitis, there is said to be no leucocytosis except in advanced pulmonary disease when it may perhaps be attributed to the suppuration. Leucocytosis does not occur in measles, typhoid fever, influenza, malaria, or peritonitis. [Other authorities find that this is true only of the tubercular variety and this is also the reviser's experience. Septic peritonitis, however, always is accompanied by leucocytosis, except in those severe cases where the organism is so overwhelmed by the disease that it makes no resistance. Absence of leucocytosis under these conditions is a grave prognostic sign.] Anæmia may also be produced by the action of certain chronic conditions which produce toxæmia, including syphilis, rickets, chronic malaria, Bright's disease, chronic suppuration, rapidly growing tumor, and certain mineral poisons, especially lead, arsenic, and mercury. In these conditions the red corpuscles are reduced in number and in size, but, as already said, the conditions as to the increase of the white cells varies.

The conditions usually classed as primary anæmia are chlorosis and pernicious anæmia.

Chlorosis, in which the characteristic alteration is a diminution in the amount of hæmoglobin without a corresponding decrease in the number of red cells, and without any considerable leucocytosis, is rare in childhood, but becomes common in girls at about the age of puberty. There is a progressive loss of strength, and increase of pallor, without wasting. The appetite is small and capricious, the bowels constipated. The greenish tint of skin to which the disease owes its name is never marked in childhood, and the face is often flushed. Deficiency in hæmoglobin renders the patient breathless on slight exertion, palpitation is easily provoked, and a systolic bruit is heard, generally at the base in the second space on the left side, but occasionally as low as the fourth space. There can be no doubt of the value of iron in the treatment of chlorosis, but when, as is the rule, constipation exists, this must be relieved, before the full effects of the remedy can be obtained. Of the preparations of iron probably the best is the perchloride, which must be given in full doses. The aloes and iron mixture (B. P.) is a very useful preparation, but as a rule salines are to be preferred as laxatives. Sulphate of magnesia may be given in solution in combination with sulphate of iron.

[Our efforts in treatment are directed towards increasing the amount of hæmoglobin, as an increase in this element is essential to restitution to health. The principal place of origin of this constituent of the blood is the intestines. Forchheimer believes we shall get our best results in treatment by giving before meals some intestinal antiseptic, such as salol or hydro-napthol, and after meals blood in one form or another. In this way the destruction of the precursors of hæmoglobin is prevented, and the hæmoglobin-producing function of the intestine is utilized to the utmost. Iron compounds may be used instead of a blood-preparation, but progress is less rapid.]

Progressive pernicious anæmia is a form of hæmolysis due usually to intestinal toxæmia; it may perhaps be caused by syphilis. An analogous condition has been traced in adults to intestinal parasites. The condition is very rare in childhood. The red corpuscles are few in number, large, some irregular in form, and some nucleated. [Megaloblasts. See Fig. 8, Plate II.] They are relatively rich in hæmoglobin; the leucocytes are as a rule not increased and may even be diminished in number. There is usually a history of long-standing gastro-intestinal derangement, and dyspepsia, vomiting, and diarrhœa are symptoms in nearly all cases. The progress of the disease is marked by a continuous increase of pallor of the skin and mucous membranes, general weakness, wasting, and loss of energy. The onset may be gradual, but the symptoms sometimes follow upon a shock. Hæmorrhages into the skin and mucous

membrane and retina ofter occur. The pulse is full and throbbing, cardiac palpitation is rare, but hæmic murmurs are heard. The symptoms are usually progressive, but intermissions may occur, and under the influence of arsenic complete recovery has taken place. In the *treatment* of the disease the administration of arsenic should be preceded and accompanied by regulation of the bowels, the use of intestinal antiseptics, and careful regulation of the diet.

Splenic Anæmia.—In infants and children up to about the age of three years, marked anæmia, attended by enlargement of the spleen, is not uncommon. Some of these cases are due to syphilis, rickets, and chronic tuberculosis ; others are examples of splenic leuchæmia ; but a considerable number remain which cannot be accounted for under any of these heads. When the patient first comes under treatment, the enlargement of the spleen is usually considerable and often enormous. It extends downwards and towards the middle line, and may occupy eventually the whole left flank, reaching beyond the umbilicus and so far downwards that the fingers can only be inserted between the iliac crest and the spleen by pressing the organ upward. The size of the organ is subject to variations, which may be, in some cases, traced to attacks of diarrhœa. The causation of the condition is obscure. Syphilis and rickets, the causes usually assigned, do not account for a large proportion of the cases, more especially those in which the anæmia and enlargement of the spleen is most pronounced. The pallor of the skin may be extreme, and it has usually a yellow tinge. There is slight œdema of the subcutaneous tissue, and the skin is dry and glazed, so that the aspect recalls that of a wax model. In some cases petechiæ appear, fade, and again appear on several occasions. There is a reduction in the number of the red cells and of the hæmoglobin ; the white cells may or may not be increased in number. Von Jaksch has proposed to make a distinct class of those cases in which considerable permanent leucocytosis is present, and has applied the term *anæmia infantum pseudoleuchæmica* to the condition. Though neither splenic anæmia nor the special form described by Von Jaksch appears to threaten life, or to be progressive in the sense in which this term is applied to pernicious anæmia, the *prognosis* is not good, general nutrition is not maintained, the child does not increase in height or weight to a natural degree, and is very apt to succumb to some inter-current disorder, especially pneumonia, measles, or an acute intestinal affection. In the treatment, attention should first be given to the gastro-intestinal disorder which is nearly always present, and in my experience little good results from the administration of iron until diarrhœa has been checked and digestion improved. The combination of the citrate of iron and ammonia (gr. iij to iv for a child of two) with a minute dose of strychnine, is to be recommended at first, followed, when improve-

ment has become established, by perchloride of iron. If there be reason to suspect syphilis, mercurials should be used in addition to the remedies already mentioned.

Leuchæmia is the term applied to conditions in which the blood contains certain abnormal white corpuscles. Two forms may be distinguished: (1) *spleno-medullary*, in which the blood contains corpuscles derived from the marrow of bone (Fig. 7, Plate II.); and (2) *lymphatic*, in which the blood contains elements derived from the lymph glands (Fig. 6, Plate II.). Leuchæmia is a disease of middle life, but may occur in infancy, and congenital cases have been described. The spleno-medullary form is less uncommon. The blood contains a great excess of white corpuscles, the most remarkable change being the presence of large mononucleated cells with granular protoplasm derived from the marrow [the so-called "myelocytes" Fig. 1, c, Plate II.]; the red corpuscles, of which some are nucleated, are reduced in number, and the hæmoglobin in greater proportion. The cause of the condition is obscure; malaria, syphilis, and injury have been assigned, and the disease has also been attributed to a specific infection. The enlargement of the spleen may be very considerable; vomiting is usually an early symptom and may be very persistent; diarrhœa may be very severe, and is sometimes due to colitis. The urine contains an excess of uric acid; the pulse is rapid and soft, and there may be a hæmic murmur. In children dyspnœa is seldom a marked symptom until the anæmia has become extreme. Petechiæ are frequent, and in some cases large ecchymoses occur. Epistaxis and bleeding from the gums are not uncommon, but hæmorrhages from other sources are rare. The retina may be the seat of inflammation secondary to extravasation, or of small leucocytal growths. In lymphatic leuchæmia there is a great increase in the number of small mononucleated leucocytes (lymphocytes) present in the blood, and there is some enlargement of the lymphatic glands. Leuchæmic patients are liable to attacks of *pyrexia* the cause of which is not explained. The **prognosis** is unfavorable, death being brought about by progressive exhaustion during one of the attacks of pyrexia, or by dyspnœa. In the **treatment**, iron, arsenic, quinine, and the inhalation of oxygen have been recommended, but more is to be expected from placing the patient under the best possible hygienic conditions, and prescribing an outdoor life.

The relation of **Hodgkin's disease** to the conditions already mentioned is not clearly defined. It is a peculiar affection of the lymphatic system, apparently ineffective, beginning locally but extending gradually to all lymphatic glands. In some cases the enlargement ensued upon a simple adenitis, but in others no such sequence of events can be traced. There is a hyperplasia of the lymphatic

glands, and nodules of lymphatic tissue may form eventually in other organs. The disease is rare, but about 16 per cent. of the cases occur in children under ten years of age. The changes in the blood are not constant; as a rule both red and white cells are diminished in number, but there may be leucocytosis. The glands most often affected at first are the cervical and those about the angle of the jaw, then the axillary. In these situations the enlargement may eventually produce very large tumors. The glands are at first distinct and easily movable, but after they have attained the size of a large almond they usually become fused together, forming large, solid, but not very hard, tumors. When this stage is reached, suppuration may occur near the surface. The inguinal, the mediastinal, and the tracheo-bronchial glands are involved usually after those of the upper part of the body. The spleen is enlarged in three-fourths of the cases, and in more than half contains lymphoid tumors, which may be present also in the liver and kidneys. Of the symptoms the most constant is fever, which, however, may be very irregular, ceasing for weeks at a time. In rare cases exacerbations and remissions alternate with regularity. Hæmic murmurs and palpitation of the heart occur in most cases, and shortness of breath may be produced, either by the anæmia or by the pressure of mediastinal glands.

The **diagnosis** from tuberculous adenitis is often difficult at first. In Hodgkin's disease the enlargement is usually more or less symmetrical, and the glands do not tend to suppurate until they have attained a large size.

[The absolute diagnosis of Hodgkin's disease is impossible without a blood count. The symptoms may be those of leukæmia and the pathology in both diseases is the same. Examination of the blood is of value negatively, in showing the absence of leukæmia. The myelocytes already mentioned as diagnostic of the *spleno-medullary* variety of the latter, are absent or present only in small numbers. The lymphocytes, greatly increased in the lymphatic type of leukæmia, are not increased in Hodgkin's disease.]

The **prognosis** is bad. As a rule the enlargement of the glands is progressive, and is attended by increasing anæmia and by dropsy, the patient becomes extremely cachectic and succumbs to exhaustion.

In the **treatment** of Hodgkin's disease the best results have been attained from arsenic in full doses, but phosphorus has also been recommended.

Hæmophilia is an hereditary disease characterized by a peculiar tendency to bleed either spontaneously or on slight injury. It affects mainly the male sex, but is transmitted by the female. Its pathology is unknown. One or more members of the same generation may be affected, but seldom all the members of the family. The degree to which the tendency to hæmorrhage is present varies in

different bleeders, and in the same person at different times. When the tendency is well marked, a slight pressure or blow is followed by considerable hæmorrhage into the cutaneous structures, and a large ecchymosis forms which passes through the regular phases of a bruise. A slight cut bleeds freely and continues to ooze for days; the mucous membranes are easily provoked to bleed, and the extraction of a tooth may be followed by serious and even fatal hæmorrhage. Bleeding from the nose is easily induced, and often difficult to arrest; while vomiting is usually attended by hæmorrhage from the stomach. Blood may appear in the urine without obvious cause. The most serious local results are seen in connection with the joints; after some slight injury or strain, sometimes without any apparent cause, blood is rapidly effused, and the joint, usually the knee, becomes distended and tender. The swelling subsides with rest, but is very apt to recur, and leads to thickening of the synovial membrane and distortion of the joint with permanent inability. In the same way, intracranial hæmorrhage may occur and may cause sudden death. The disease usually manifests itself in the first year of life, sometimes about the second dentition. The prognosis as to ultimate survival is bad, as many of the patients succumb during childhood to some intercurrent malady which is aggravated by the tendency to bleed.

In the **treatment** the main indication is to protect the patients from injury. Since the hæmorrhage is almost exclusively capillary, ergot and other similar drugs are not likely to be of much service for the arrest of hæmorrhage; for this purpose pressure at the bleeding point is most to be trusted, and Wright recommends the use of a tampon soaked in a one-per-cent. solution of calcium chloride, which produces a very firm clot.

CHAPTER XXII.

DISEASES OF THE THYROID AND THYMUS GLANDS.

Acute Thyroiditis—Goitre—Cretinism—The Thymus Gland; Anatomy; Thymic Asthma.

THE THYROID GLAND.

Acute thyroiditis is a very rare affection. It has been observed chiefly about the age of puberty, but has also been met with in young children.[1] It causes enlargement of the gland, which forms a swelling on each side of the neck and extending across the middle line. The swelling moves with deglutition, and is thus distinguished from that produced by lymphadenitis or subcutaneous phlegmon. The inflammation of the thyroid is accompanied by fever and causes some dysphagia, pain on movement, tenderness, enlargement of the veins of the neck, and slight cyanosis. The amount of dyspnœa varies; in some cases it has been considerable, causing the head to be retracted. In most cases the swelling begins to subside in a few days, though some enlargement and hardness of the gland may persist for weeks. Such cases may, perhaps, be rheumatic in nature. In other cases, generally those in which the patient has been suffering from erysipelas, suppuration has occurred; it is an occasional complication of typhus fever and pyæmia, and has also followed thyroiditis due to injury. The treatment of acute simple thyroiditis must be conducted on general principles; if the enlargement of the gland be sufficiently great to produce symptoms of pressure on the larynx, leeches should be applied. The dyspnœa has, in some cases, been so severe as to render tracheotomy, or division of the thyroid isthmus, imperative.

Goitre may be congenital, and is then due to hyperplasia of glandular and interstitial substance. It is seldom met with except in goitrous localities. At a later age cystic bronchocele is not very uncommon in certain districts. **Exophthalmic goitre** has been observed in childhood, but is an exceeding rare affection at that age.

Cretinism is a condition of defective development of body and

[1] Barlow (*Trans. Clin. Soc.*, vol. xxi., p. 67) has recorded a case in a boy aged 3 years. His paper contains an analysis of the literature.

mind due to the absence of the thyroid secretion, owing to want of development of the gland or to its atrophy in early life.

Sporadic cases occur in most civilized countries, and are not uncommon in London. The degree to which the arrest of development is carried, and to which the special characters are produced, varies in different cases in dependence, probably, on the date at which the atrophy of the gland occurs and the degree to which it is carried. The body is dwarfed, and the mind also. The appearance is characteristic. The face is broad and pale; the lips heavy; the eyelids thick, so that the palpebral aperture is narrow. The

FIG. 24.

A B

Acquired cretinism. A, The patient is stated to have developed normally until the age of 5 years, when she had a severe attack of scarlet fever, after which growth nearly ceased, and a great change came over her mental state. At the age of 18½ years, when the photograph was taken for me by Dr. George W. B. Waters, she was childish, showed no signs of puberty, and was slow in movements and in speech. Her height was 44 inches, and her weight 48 lbs. The average height of a girl of 18 years is 62.5 inches, and the average weight 117.7 lbs. The average height of a girl aged 5 to 6 years is 40.8 inches, the average weight 39.6 lbs. (see page 3). B, The same patient four months later, showing some improvement in the intelligence of her expression—there had also been some increase in height—after treatment by thyroid extract.

tongue is large, and may be so much hypertrophied (macroglossia) that it cannot be withdrawn between the lips; the body is clumsy; the hands are broad and fin-like; the feet large and flat; the skin is dry and harsh; the hair thin and lustreless. Above and immediately below the clavicles there are, in many cases, masses of subcutaneous fat. The gait is clumsy, and the movements of the

hands slow and awkward. The extremities are cold. The intelligence and general condition of the patients are worse in winter. The arrest of physical and mental development may be such that a cretin of twenty years is no bigger than a child of three, is an idiot and unable to speak, and must be fed and dressed. In milder cases growth of body and mind is retarded, but not completely arrested.

Fig. 25.

Congenital cretinism, showing an extreme degree of the condition. The patient was a girl aged 18 years at the time the photograph was taken; the stupid (imbecile) expression, the large tongue, clavicular masses of fat, and general stunting of growth are well seen.

A cretinous girl of nineteen, for instance, had the aspect of a dull child of nine or ten; she stood 3 ft. 8 in., could speak in a slow, monotonous voice, and could do a little house work (Fig. 24). The age at which the defect in the bodily and mental development of the child is first noticed varies, but the aspect may be quite charac-

teristic before or soon after the child has completed its first year (Fig. 25).

The *diagnosis*, owing to the dwarfing of the body, the condition of the skin, the cervical lipomata, and the aspect of the face, is

Fig. 26.

Congenital cretinism. The same patient as Fig. 25 after treatment for six months with thyroid extract. (Dr. W. Rushton Parker's case; the history of the patient is related in the *Brit. Med. Journ.*, 1896, vol. i., 1350.)

usually easy; but in infancy and early childhood it may sometimes be difficult to feel certain that the mental deficiency may not own another cause. The type of idiocy commonly spoken of as Mongolian, illustrated in Chapter XLI., presents a considerable resemblance to cretinism. Growth is stunted, the expression dull,

the mouth open, the tongue often large, the movements uncertain, and speech, which is acquired late, is monosyllabic. The skin is coarse, the subcutaneous tissues are thick, and marginal blepharitis

FIG. 27.

"Adolescent" cretin, aged 22 years, after treatment for three and a half years with thyroid extract, showing bending of leg-bones, comparative growth of arms and legs, and alteration in shape of hands. (From a photograph by permission of Dr. John Thomson.)

is very common. Lipomata do not occur, the slowness of intellect in infancy is less marked, movements are usually quick and jerky, and bodily development though slow is not arrested as in well-marked congenital cretinism.

[The condition known as fœtal rickets achondroplasia or the chondrodystrophia fœtalis, is liable to be mistaken for cretinism. Osler[1] reports the following 2 cases:

"The parents were healthy French Canadians. There were fourteen children in the family, the eldest twenty-seven, the youngest four. Five children had died in infancy. With the exception of the dwarfs, the children were all very healthy and well grown.

"Wilhelmine C., aged sixteen years, height 86.5 cm. (34 inches) (Fig. 28). The mother did not remember anything abnormal about her as a young infant. She walked when eighteen months old. The head seemed large, and the mother said that the fontanelle did not close until the sixth year. When between three and four it was noticed that she did not develop naturally, and that the joints were very large. She is bright looking and intelligent, but somewhat full and coarse-featured. The head measures 56 cm. The

Fig. 28.

Fig. 29.

teeth are well formed. She talks fluently and well, and has learned to read a little, and is beginning to write, but she is backward for a girl of her age. The most remarkable phenomenon is the condition of the joints of the long bones. The shafts are short and look thin, and the articulations are very large and irregular. The shoulders are not much affected, but the elbow-joints, the wrist-joints, and the knees and ankles are enormously enlarged. She is

[1] Osler: Sporadic Cretinism in America. (*The American Journal of the Medical Sciences*, Vol. CXIV., No. 4.)

a little knock-kneed when she stands. The mobility in the joints is perfect.

"Alphonse C., aged eleven and a half years, height 87 cm. (33½ inches) (Fig. 29). The mother did not notice anything special about him except that he was late in walking, and the anterior fontanelle did not close until between the third and fourth years. He did not seem to grow much after the fourth year. He presents an identical picture to that of his sister. His head is large, but well formed. He is very intelligent looking and bright, and is good tempered. The articulations are extraordinarily large, and contrast with the smallness and shortness of the shafts of the bones. He is somewhat pigeon-breasted, and when he stands is knock-kneed.

"The trunk and head in both these children look almost of natural size, but the shortness of the legs, and particularly the shortness of the shafts of the long bones, is very striking. The thyroid gland was not enlarged in either case.

"The relation of this remarkable condition to cretinism is very carefully discussed by Kaufmann,[1] and more recently by Bircher,[2] to whose papers the reader is referred. John Thomson, in the *Edinburgh Medical Journal* for 1893, gives excellent illustrations of the adult form.[3] The thyroid is not usually involved, though it has been found absent in a fœtus which presented this condition (Bowlby).[4] The intelligence is not specially disturbed, the facial and cranial characters are not those of cretinism, and myxœdema is not present. The most characteristic feature is the dwarfing, with remarkable shortness of the limbs (micromelia), owing to disturbance of the growth of the shafts of the long bones, and with, in most cases, enormous enlargement of the articulations, due to a hyperplasia of the cartilaginous ends of the bones. Bircher concludes that the condition is quite independent of the state of the thyroid gland. He is in error, however, when he states that the cases of sporadic cretinism described by Cushing and Fagge belong to this group."]

Treatment by the administration of thyroid gland should be resorted to in every case in which any doubt exists, since it produces distinct amelioration in cretinism, and slight improvement in Mongolian idiocy. The experience of most observers probably agrees with that of Byron Bramwell,[5] who points out that most cases of sporadic cretinism are very susceptible to the action of thyroid extract. A small

[1] Untersuchungen die über sogenannte fœtale Rachitis (Chondrodystrophia Fœtalis). Berlin, 1892.

[2] Lubarsch und Ostertag, Ergebuisse, Abt. i., 1896.

[3] They are reproduced in Gould and Pyle's "Anomalies," etc.

[4] Pathological Society Transactions, 1884.

[5] *Edinburgh Hospital Reports*, vol. iii., p. 116.

dose should be administered at first, gr. j to ij (a quarter of an average sheep's thyroid). In a cretin even this small dose may produce a series of symptoms to which the term *acute thyroidism* has been applied—gastro-intestinal disturbance (furred tongue, vomiting, diarrhœa), prostration, sweating, headache, myalgia, flushing, and feelings of discomfort. The patient should be kept at rest, and even in bed for the first week of the treatment, but after a time the susceptibility diminishes, and the dose may be increased cautiously until 5 grains are taken daily. The improvement in the general, but especially in the mental, condition is very remarkable, though the change is less rapid and striking than in the myxœdema of adults (Fig. 26). The treatment must be continuous, but after the maximum dose has been taken for two or three months, it may be reduced, the condition of the patient being carefully watched for any signs of deterioration. How far a cretin may be brought towards a normal standard of development cannot at present be stated; but experience seems to show that relatively slight improvement only must be looked for, unless the treatment be commenced when the child is yet young. Early diagnosis therefore is a matter of great importance. In cretins, especially those in whom a certain amount of development occurs during the early years of life, lateral curvature is not uncommonly observed, and one of the most disagreeable results of thyroid treatment

Fig. 30.

Fig. 31.

is that during the process of growth which it stimulates, this lateral curvature is very apt to increase and to cause much aching pain in the part. This may be to some extent combated by gymnastic exercises, but other bony deformities may occur, due to thickening and

softening—apparently of a rickety nature—of the bones of the extremities[1] (Fig. 27). It may therefore be necessary to keep the patient recumbent, and even to apply splints for some months, while the effect of the treatment by thyroid is producing its maximum effect.

[Osler's[2] series of cretins illustrate so well the characteristic appearance of the condition and especially the effects of thyroid treatment that it is reproduced here. "Fig. 30 presents a typical picture of a sporadic cretin, aged seventeen years, under the care of Dr. J. C. Carson, of Syracuse, New York. Fig. 30 was taken a year before treatment and Fig. 31 illustrates the condition a year after."

Fig. 32 represents a girl, Theophilia P., aged two years, admitted to the Johns Hopkins Hospital, December 18, 1895. Mother a

Fig. 32.

Fig. 33.

Pole, no goitre in the family. Child presents the typical aspect of infantile myxœdema. The thyroid gland can be felt and seems a little large. She can neither sit nor stand; is pale, the skin of the face having a sallow waxy appearance. Tongue is large and held out all the time. The expression of the face is dull and idiotic. Puffiness and myxœdematous character of the skin of both hands

[1] See a paper by Dr. John Thomson, *Brit. Med. Jour.*, 1896, vol. ii., p. 618, and the discussion thereon.

[2] Osler: loc. cit.

and face. The head measures 45 cm. in circumference, 25½ cm. in the transverse, and 35 cm. in the antero-posterior. The anterior fontanelle is large, almost as much so as at birth. Child's

FIG. 34.

FIG. 35.

length on admission was 65 cm. The blood-count gave 4,648,000 red corpuscles, 80 per cent. of hæmoglobin, and about 11,000 leucocytes.

FIG. 36.

FIG. 37.

On December 20th she was placed on the thyroid extract prepared by Dr. Abel. On beginning the treatment she weighed twenty pounds. Within two months, as illustrated in Fig. 33, the condition

improved remarkably. She took an interest in surrounding objects. The eyes had become bright and intelligent; the puffiness of the face and hands had almost wholly gone. As shown in the photo-

Fig. 38.

Fig. 39.

graph, the palpebral orifices had become much larger. The tongue was not held out so far from the mouth, and the drooling had almost ceased. She remained in the hospital until September 1, 1896. The myxœdematous condition disappeared entirely, she gained eight

Fig. 40.

Fig. 41.

pounds in weight, and had thriven in every way. It is interesting to note in this case that from March 20th to April 4th, the child received no thyroid extract. She had had a little fever and bronchitis,

and it was stopped. On the latter date it was noticed that the skin had become harsh and dry, the eyelids more puffy, and the child did not seem nearly so well.

"Fig. 34 shows the patient of Dr. Elsner, of Syracuse, New York. The child was eighteen months old at the beginning of treatment, and the photograph shows a very characteristic state of infantile myxœdema. Fig. 35 shows the state thirteen months after treatment."

"Figs. 36 and 37 illustrate the case of Dr. Vinke, of St. Charles, Mo., a boy, aged six years. Fig. 37 shows the condition five months after treatment. In a year and a half he grew nine inches."

FIG. 42.

FIG. 43.

"Figs. 38 and 39 show a patient of Dr. Dickson L. Moore, of Columbus, Ohio, a girl, aged nine years. The treatment was begun August 12, 1896. Fig. 39 shows the condition seven months later, March 20, 1897. The child had gained four inches in height, and the entire appearance had changed remarkably."

"Figs. 40 and 41 show a sporadic cretin at the age of thirty years, patient of Dr. Sinkler. The height was $112\frac{3}{4}$ cm. Fig. 41 shows the condition a year after treatment. She had grown nearly 7 cm., and had lost much of the myxœdematous characters. This

case is of special interest as showing the importance of the treatment even in adults."

"I know of no single set of photographs which show in quite the same way the phenomenal change as in this series of pictures very kindly sent by Dr. Coyner, of Peoria, Ill.

"Figs. 42 and 43 show the very characteristic appearance of a sporadic cretin, aged twenty-three months, length 28 inches, circumference of the abdomen 19 inches. Fig. 44 shows the change after three months' treatment; the abdomen measured 16 inches. Fig. 45 illus-

FIG. 44.

FIG. 45.

trates the condition after five and a half months' treatment; height 30 inches; abdomen measured 15 inches. Fig. 46 shows the change after seven and a half months' treatment; while the last picture, Fig. 47, shows eleven months after beginning the use of the thyroid a perfectly-natural looking child."]

The Thymus Gland.

The large size of the thymus gland at birth is one of the most striking features of the infantile thorax.[1]

[1] Ballantyne, "Introd. to the Diseases of Infancy," Edinburgh, 1891, p. 64.

It occupies the anterior mediastinum, lying behind the manubrium and the upper part of the body of the sternum, and the three upper costal cartilages. Its upper border projects above the suprasternal notch, and almost touches the isthmus of the thyroid. It varies a good deal in size at birth, but the maximum measurements are about

Fig. 46.

Fig. 47.

as follows:—breadth, 3.5 cm. ($1\frac{1}{3}$ in.); length, 5 cm. (2 in. nearly); thickness, 2.5 cm. (1 in. nearly). It begins to atrophy about the end of the second year, undergoing fatty and fibrous changes which are usually complete about the tenth year. In infancy it produces an area of diminished resonance on percussion which may extend as low as the level of the third rib.

Neither the functions nor the pathology of the gland are well understood; in syphilis it may be the seat of multiple abscesses, and tuberculosis of other organs may be accompanied by caseous masses in the thymus. It has long been supposed that the gland, if enlarged,

might produce dyspnœa, and attacks attributed to this cause have been called **thymic asthma**. Though this view has been controverted, there can now be little doubt that enlargement—it appears to be due to general hyperplasia—may occur in infancy and early childhood. The enlargement makes itself evident by producing severe attacks of dyspnœa, due probably to sudden hyperæmia of the glands In some cases the first attack of dyspnœa is fatal. The child in the midst of apparent health, sometimes on awakening from a sound and natural sleep, is seized with the most intense dyspnœa, and dies asphyxiated before assistance can be rendered. In other cases dyspnœa develops more gradually and becomes constant, though liable to temporary aggravation. In such cases tracheotomy has sometimes failed to give relief until a tube of unusual length has been inserted into the trachea.

CHAPTER XXIII.

DISEASES OF THE HEART.

[Normal Position of the Heart]—Congenital Affections of the Heart—Pericarditis—Pleuro-pericarditis—Acute Endocarditis; Simple; Malignant—Chronic Endocarditis—Valvular Disease.

[**Normal Condition of the Heart in Infancy and Childhood.**—A knowledge of the position of the heart and of its relation to the chest-wall in infancy and childhood is essential to a thorough grasp of the abnormal condition of this organ. In infancy the heart is ordinarily more horizontally placed than it is in the adult; hence we see the apex-beat higher and further to the left. With the growth of the child, this relation of apex-beat to chest-wall varies, owing partly to changes in position of the heart itself. As a result, the apex-beat swings downwards and inwards and consequently will be found in different positions at different ages. Thus up to about the third or fourth year, it is outside the mamillary line, and in the fourth space.

From the fourth to the eighth or ninth years it is in or near the mamillary line and generally in the fourth space, but occasionally in the fifth. About the eleventh to the thirteenth year we find it in the adult position, the fifth space quarter to half an inch inside the nipple-line.

The upper limit of "absolute dulness" in infancy is at the fourth rib; at six years is at the lower border of the third rib, and at ten or twelve years the upper border of the third rib, or sometimes as high as the second intercostal space. The right limit of dulness is the left para-sternal line.

It is important to bear in mind these different boundaries, as failures so to do may lead to erroneous deductions from the results of physical examination of the chest. It is also important to remember the thinness of the chest-wall in childhood and the consequent ease with which sounds are diffused throughout the thorax.]

Congenital affections of the heart are due to defects of development, to fœtal endocarditis, or to a combination of these two conditions. The more extreme deformities are incompatible with extra-uterine life.

The changes in the valves produced by fœtal endocarditis are usually

indurative, the valves being thickened, irregular at the edges, and sometimes adherent. Thus two of the pulmonary valves may be adherent, so that the orifice has but two valves, or all three valves may be so welded together as to form a diaphragm with a single aperture. The auriculo-ventricular valves may be distorted and adherent to each other, and the chordæ tendineæ thickened and shortened. Fœtal endocarditis affects usually the right side.

The abnormalities compatible with life for at least some years may be divided into those affecting the septa and the orifices.

The *auricular septum* may be defective owing to (1) the existence of minute perforations in the valve of the foramen ovale, or (2) to the failure of this valve to become attached along the whole of its margin, so that a slit is left. Neither of these conditions necessarily interferes with the cardiac function. When the defect in the foramen ovale is considerable, a loud systolic murmur is produced, which is heard best at the base of the heart in front, but is audible also at the back. Children with this defect are very liable to bronchitis, which is attended by much embarrassment of the circulation. A defect in the *ventricular septum* is associated usually with some other congenital abnormality, especially obstruction at the pulmonary orifice. The defect occurs usually at the base in the "undefended spot," the membranous space between the mitral and tricuspid valves. This defect produces a loud murmur replacing the first sound, heard best over the lower part of the sternum, but also in the axilla and back.

Abnormalities at the *pulmonary orifice* constitute 86 per cent. of all cases of congenital heart disease which survive beyond the age of twelve years. The commonest condition is stenosis, produced by a blending of the valves into a single membrane with an orifice which may be very small. In some cases there is obvious evidence of endocarditis, but in others the diaphragm is smooth and thin, the abnormality being due, apparently, to a developmental defect. It is associated usually with imperfect closure of the ventricular septum; the condition is not incompatible with survival to adult age. The physical signs produced are a loud systolic murmur and feeble second sound in the pulmonary area; but if the ventricular septum be also deficient a systolic bruit is heard at the lower part of the sternum. A more serious grade of pulmonary obstruction occurs when there is also narrowing of the *conus arteriosus* of the right ventricle, which is associated with imperfection of the ventricular septum and patency of the foramen ovale and ductus arteriosus. Finally, associated with the defects just enumerated, there may be complete obliteration of the pulmonary orifice, a condition incompatible with long life.

Congenital affections of the *aortic orifice* are rare. The commonest form is that in which the number of valves is reduced to two by a blending of two valves, due to endocarditis shortly before birth.

Possibly in some instances, the defect is produced by developmental abnormality, but in either alternative the valves are very liable to become the seat of endocarditis after birth.

Of the *symptoms* of congenital heart disease, the most striking is cyanosis, whence the name **morbus cæruleus** applied to the condition. It is absent in only 10 per cent. of all cases, but its intensity varies and it is always increased by crying or exertion. Lividity is noticed usually during the first few weeks of life. The nose, ears, lips, fingers, and toes have a purplish tinge, while the rest of the surface has a dusky tint. Cyanosis is most marked and most extensive in obliteration of the pulmonary orifice with patent foramen ovale. It is due to deficient aëration of the blood, which contains a very high proportion of red cells. Children presenting cyanosis do not thrive well, and are usually backward in intelligence. They feel the cold much, are very liable to bronchitis, and after a time the fingers and toes become clubbed. There is always some dyspnœa on exertion. They are liable also to attacks of dyspnœa without apparent cause. Death may ensue in one of these attacks. More often it is brought about by bronchitis; but it should be noticed that a considerable number of cases die of intracranial abscess.

The **diagnosis** of congenital heart disease is usually easy, owing to the co-existence of cyanosis and cardiac murmur. However loud the murmur, there is no thrill. In doubtful cases considerable increase of dulness to the right without signs of cardiac failure would favor the diagnosis of congenital disorder. Patent ductus arteriosus causes a loud vibrating systolic bruit, best heard over the upper part of the sternum. It is not accompanied by hypertrophy of the left ventricle, as is disease of the aortic orifice, but patency of the ventricular system may be attended by considerable hypertrophy of the left ventricle. In congenital heart disease without urgent symptoms **treatment** can do little beyond guarding the patient against bronchitis, and placing him in the best hygienic conditions obtainable. It should be borne in mind also that children with congenital defects at the pulmonary orifice appear to be particularly liable to pulmonary tuberculosis. Cyanosis and dyspnœa will be relieved by the use of saline laxatives, and in the severe dyspnœal attacks venesection is justifiable. The routine use of digitalis is to be condemned, but it may be valuable when there are signs of cardiac failure.

Pericarditis.—Inflammation of the pericardium is usually secondary to, or a part of a more widespread infective process. The commonest cause in children over three years of age is the rheumatic state of which pericarditis may be the first, or, at a particular time, the only manifestation. It occurs in about one-fourth of the fatal cases of chorea. It may be a complication of various acute infectious fevers, especially scarlet fever, but also, though more rarely, of

measles, small-pox, influenza, diphtheria, and enteric fever. It may be produced also by tuberculosis, by septicæmia, and in the course of acute Bright's disease. It may be determined by extension from pleuro-pneumonia, and this is probably the commonest cause in children under three years, though septic pericarditis associated with inflammation of the navel occurs sometimes in infants during the first weeks of life. Its occurrence before birth has been recorded. Acute pericarditis is attended by the formation of fibrinous exudation, but the amount of fluid effused varies greatly. There may be little or no excess of fluid in the pericardial cavity, and the quantity of plastic material on the surface varies from an amount sufficient only to produce a dulling of the serous surface to a thick membrane which has a corrugated or shaggy appearance. The pericarditis due to acute rheumatism is generally attended by effusion, the amount of which may be very considerable. In rheumatism, and in tuberculosis, and septicæmia at first, it is serous, but often contains shreds of fibrin which may form a thick layer on the serous surface. It contains, sometimes, a larger number of corpuscles, and is then opalescent or sero-purulent. Tuberculosis produces in time a ragged gray or yellowish false membrane, with thick, creamy pus in the pericardial cavity. In severe cases of pericarditis the myocardium is inflamed for a varying depth, and endocarditis is a common accompaniment, though seldom due to direct extension.

The **symptoms** are often very indefinite, especially in young children. There is usually pain referred to the præcordia or to the epigastrium, and there may be some tenderness in these situations. When pericarditis occurs as a primary affection its onset is attended usually by chilliness, or shivering, and some elevation of temperature, but it may be very insidious. The child is noticed to be indisposed to play, short of breath and pale for a week or two before it is brought for treatment. Then, perhaps, a large effusion is found, or a characteristic friction sound. Considerable effusion with a very insidious onset should lead to a suspicion of tuberculosis. In the dry form the physical signs are those produced by the rubbing together of the roughened pericardial surfaces. Fremitus may be felt over the right ventricle, and on auscultation a double to-and-fro friction sound. This corresponds with the systole and diastole of the heart, and is rough and grating, or resembles that produced by new leather. It is audible most often at the base or in the fourth space (over the right ventricle), more rarely at the apex. It is heard usually over a very limited area, but may be conducted for some distance down the sternum. It is not constant in intensity, and may be abolished by effusion. In dry pleurisy also it may disappear owing, possibly, to the formations of adhesions, but it reappears if the inflammation extends.

The **diagnosis**, when pericarditis occurs as a complication of acute rheumatism while the patient is under observation, is comparatively easy, since the changes in the physical signs are characteristic. But in cases with an insidious onset it may be difficult. Thus friction at the base may suggest a diagnosis of aortic incompetence, but the limited area over which the double murmur is heard is peculiar, and the harsh rubbing character and the absence of the characteristic modification of the pulse will generally permit a diagnosis to be made with confidence. Friction is occasionally due to pleuro-pericarditis (see below). Dry or plastic pleurisy may be succeeded by effusion, or it may end in the production of more or less extensive adhesions.

If pericarditis produces considerable effusion, the symptoms and physical signs are much more distinctive. There is usually a good deal of pain referred often to the epigastrium and aggravated by pressure. The face is anxious and dusky, the patient lies on the left side, or sits up, and is breathless on slight exertion. The pulse is rapid, and at the wrist may be very weak or disappear altogether with each inspiration—*pulsus paradoxus*. Cough is often troublesome, and dysphagia may be present. These symptoms are to be attributed to the embarrassment of the circulation, and are attended usually by nervous depression, which, in later stages, gives way to restlessness and insomnia, to delirium, and, finally, to coma. The physical signs vary with the degree of effusion—bulging of the præcordia, fulness of the intercostal spaces, and œdema of the skin may be marked in children, even at an early stage ; and after a time considerable enlargement of the lower part of the front of the chest on the left side may be produced. Expansion of the left side is diminished owing to compression of the left lung. The apex-beat is displaced upwards and outwards, and becomes weaker and finally disappears as the effusion progresses, though it may generally be perceived if the patient bends forward or lies on the face. On percussion the area of dulness is increased, both upwards and over the sternum. The dull area has an irregular pyramidal shape. Much importance has been attributed to disappearance of resonance in the fifth interspace on the right side ; this may occur even early in pericarditis with effusion. It may also be observed in dilatation of the right ventricle. The dulness may extend upward into the second and even into the first interspace on the left side. The upper limit may vary from time to time, the variations depending, in part, on the quantity of fluid, but probably, in part, also, on the degree to which the pericardial sac gives way under the pressure.

Displacement and depression of the left lung cause the percussion note in the axilla below the nipple line to become flat or tympanitic. As the amount of fluid increases, the to-and-fro friction sound diminishes and may eventually disappear ; on the other hand, it may

persist at the base, so that the existence of friction does not disprove the presence of a large amount of fluid. The heart sounds grow weaker and more distant as effusion increases. The course of the affection is very variable; rapid effusion is often followed by rapid absorption. In septicæmic pericarditis pus is rapidly formed, and early death is the rule. In the more chronic cases the pus may point near the sternum.

The **diagnosis** of effusion may be difficult if the case cannot be watched from the first. If very large, it may be mistaken for effusion into the left pleura, but the situation of the dulness and the muffled character of the heart sounds, as well as the position and the character of the apex-beat, will generally prevent error. From dilatation of the heart the diagnosis of effusion into the pericardium may be very difficult, and mistakes have been made by the most careful. In dilatation the impulse is wavy, and visible usually in several spaces; the shock is more distinct; the sounds clearer, being often very sharp and ringing; the area of dulness is not pyramidal, and does not rise above the third space; and there is no tympanitic note in the axilla.

Adhesion of the two surfaces of the pericardium is a common consequence of pericarditis. Rheumatic pericarditis may lead to a few scattered adhesions, especially over the right ventricle, or to more extensive and even universal adhesions. In adherent pericardium from this cause the thickening is not great, and the adhesions are fibrous; but in tuberculous pericarditis there may be great thickening with caseous nodules in the substance. Small, limited adhesions give rise to no symptoms or physical signs. When extensive or universal, more or less cardiac hypertrophy ensues. There is often considerable bulging on the left side in front, so that the deformity of the chest is obvious at the first glance. The area over which the cardiac impulse is felt extends downwards to the sixth space and outwards beyond the nipple. The maximum impulse is usually a good deal to the right of the apex, and a characteristic sign when present is a retraction at the apex or lower sternal region with systole, and a rapid rebound during diastole (diastolic shock, with collapse of the cervical veins).

[Another sign which according to Osler is of diagnostic value is that known as "Broadbent's sign." This consists in a systolic retraction in the line of attachment of the diaphragm from behind, in well-marked instances seen on both sides, often only on one side and in only one interspace, the 10th or 11th. Broadbent attributes this sign to broad adhesions between the right ventricle and the diaphragm as a result of which there is a systolic tugging at the diaphragm with a consequent retraction of the chest-wall.]

The area of absolute dulness is usually considerably increased.

There may be no murmur, though a systolic bruit at the apex is the rule.

In the **treatment** of acute pericarditis the most important element is rest in bed. When the symptoms are severe, an ice-bag should be applied over the præcordium. It should be used on the first occasion for about an hour, then for two hours, and, finally, should the symptoms persist, it may be applied continuously. For the relief of pain and irritability morphia may be necessary. In less severe cases dry or, in robust children, wet cupping may be sufficient. A small mustard poultice will often have the same effect, but there is the objection to its use that it interferes with subsequent applications, and hot fomentations or poultices are preferable, especially in young children. In rheumatic cases the salicylates will be used, but their influence over pericarditis is not very marked. It is doubtful whether potassium iodide hastens the absorption of fluid, and its depressing effect is undesirable. With rest and careful nursing the fluid, as a rule, disappears spontaneously. When a rapid and copious effusion is threatening life, it is justifiable to tap the pericardial sac. The point selected should be the fourth space, one inch to the left of the sternal margin, or in the fifth space a little farther out. [Rotch's researches have shown the greater safety of tapping in the fifth right interspace. We thus avoid the heart and are sure of striking the fluid even though it be small in amount.] If the effusion be very large, it has been recommended to insert the needle in the costo-xiphoid angle, close to the costal margin, and to push it upwards and backwards. If the fluid be purulent, incision and drainage appear to be the most rational treatment.

Ashby has applied the term **pleuro-pericarditis** to a rare condition, the exact nature of which is somewhat obscure. As Osler has observed: "In children chronic adhesive pericarditis may be associated with proliferative peritonitis, perihepatitis, and perisplenitis, in which condition ascites may recur for months, or even for years." Ashby describes such a sequence of events as a consequence of inflammation of the serous membrane reflected over the anterior edges of the lungs, the only definite sign being a friction sound, synchronous with the cardiac beats, more intense during inspiration, and disappearing during expiration. Finally the edge of the lung becomes adherent to the pericardium. A subacute inflammatory process, which is in some cases tuberculous, may ensue in the mediastinum (mediastino-pericarditis). The pressure upon the veins entering the chest leads to secondary fibrosis of the liver, and chronic ascites may be the most prominent symptom. The patient may live for long—until the portal obstruction becomes too great to be compatible with life.

Endocarditis.—Inflammation of the lining membrane of the

heart is confined usually to the valves. It may be acute, with the production of vegetations, or loss of substance by ulceration, or chronic, and attended by sclerosis, with thickening and puckering.

Two forms of acute endocarditis may be distinguished—simple and malignant—which differ in the degree but not in the nature of the anatomical lesions.

Acute Simple Endocarditis may occur at any age, and even before birth, but it is very uncommon during the first two or three years of life. As in the adult, so in children simple endocarditis is more often due to the rheumatic poison than to any other cause. It may be at the time of its occurrence the only manifestation of the rheumatic state. Sometimes it occurs as a complication of erythema nodosum, peliosis rheumatica or acute tonsillitis, but usually it comes on during an attack of acute or subacute rheumatic arthritis. It is a not uncommon complication of scarlet fever, with or without arthritis, and of pneumonia. It is one of the rare complications of measles, small-pox, chicken-pox, diphtheria, and enteric fever. It is often present in chorea, and is found after death in the majority of all fatal cases. Recurrent or relapsing endocarditis—that is to say, acute inflammation grafted on the sclerotic condition produced by chronic or sclerosing endocarditis—yields perhaps the largest number of cases actually met with in practice.

Malignant Endocarditis seldom, if ever, occurs as a primary disease in children. In them it is secondary most often to recurrent endocarditis, but it may occur as a complication or sequela of various acute diseases, especially pneumonia, but also of rheumatism, septicæmia, erysipelas, gonorrhœa, and of acute osteo-myelitis or arthritis. It is said to be rare in chorea, but the most typical cases I have seen in children have been in sufferers from chronic relapsing endocarditis which originated during an attack of chorea.

Morbid Anatomy.—Acute endocarditis is characterized by the production of vegetations on the valves, especially at their edges. They consist of granulation tissue, and have an irregular fissured surface coated with fibrin, in which micro-organisms may be demonstrable. The vegetations (1) may undergo resolution, a small nodular thickening of the valve remaining; or (2) may increase in size, and then undergo disintegration and ulceration (malignant endocarditis). The ulcerative process begins in the vegetation, but may extend to the endocardium, producing a more or less extensive necrosis, which may cause perforation of a valve, of the septum, or even of the heart; or suppuration may occur at the base of the vegetations, with the production of small abscesses. The vegetations and ulcers contain various microbes—generally the *streptococcus pyogenes*, or one of the *staphylococci;* but the bacillus of typhoid, anthrax, and tubercle, the *gonococcus* and the *micrococcus lanceolatus*,

have also been met with. Except in fœtal life, the left side of the heart is that usually affected, and when the right side is attacked it is due to secondary infection. Portions of the vegetations or of their fibrinous caps may be detached, forming emboli, which may become impacted in the spleen, kidneys, brain, intestines, or other organs. In malignant endocarditis these emboli, being infective, give rise to abscesses, and in some cases a very large number of minute abscesses may thus be produced.

The **symptoms** of simple endocarditis are slight pyrexia, with increased rapidity and sometimes irregularity of the heart or palpitation. They coincide as a rule with the development of a soft murmur, usually at the apex which amounts at first to no more than a roughening of the first sound. Since, in the vast majority of cases, endocarditis occurs first during an attack of acute rheumatism or other acute disease, in the course of which a *bruit de souffle* may be produced without endocarditis, it is obvious that the diagnosis must often be conjectural. The course of simple endocarditis is usually short, from a few days to a week or two. It may terminate in apparent recovery, but in a large proportion of cases more or less chronic sclerosing endocarditis ensues.

The symptoms produced by malignant endocarditis, whether it be a complication of an acute infectious disease or secondary to chronic endocarditis, are usually of the typhoid type—prostration, somnolence, muttering delirium, severe sweats, sometimes rigors, irregular temperature, and petechiæ or septic rashes. In a minority of cases the symptoms are distinctly pyæmic, with marked rigors, profuse sweating, septic rashes, diarrhœa, and often jaundice. In either form the sudden onset of pain in the left side, the appearance of blood in the urine, or the development of hemiplegia may indicate the occurrence of embolism in the spleen, kidneys, or brain. The course of malignant endocarditis may be short, not more than a few days; usually it is prolonged, especially in the cases which ensue upon recurrent endocarditis, and there may then be many remissions, followed by relapses.

The **diagnosis** of malignant endocarditis is often difficult. When in the course of endocarditis the general constitutional symptoms become severe, with an irregular temperature, especially if rigors occur, the supervention of malignant endocarditis may be assumed, and the diagnosis will be placed beyond doubt by the occurrence of suppuration in internal organs. Cases liable to be mistaken for typhoid fever occur in children probably less often than in adults; distinctive points are the more abrupt onset, often with pain referred to the cardiac region, the irregular pyrexia from the first, rigors, and the early occurrence of dyspnœa.

In the **treatment** of simple endocarditis, the most important indica-

tion is rest in the recumbent attitude, and it is very doubtful whether in cases of moderate severity, treatment by drugs has any beneficial effect upon the course of the disease. The salicylates appear to exercise very little influence, and I have never seen any advantage result from the use of aconite. In severe cases the ice-bag may be of use. Small doses of opium relieve pain and quiet irregular cardiac action, and at later stages, if there be signs of cardiac failure, digitalis may be tried cautiously. If the endocarditis be complicated by pericarditis, the treatment should be directed to the relief of that condition. When there is much excitement potassium bromide is the most innocuous sedative. The diet should be light, and should consist chiefly of milk diluted with some slightly aërated water, given in small quantities. At the commencement of treatment the bowels should be unloaded, and for this purpose calomel is probably the best drug. Flatulent distension of the stomach or colon should be watched for and treated at once, since the embarrassment of the heart may thus be greatly increased. After recovery from the acute attack, great care is required for a long period. The child should be carefully clothed and dieted, and a boy ought not to go to a public school for at least a year.

In malignant endocarditis very little can be done, and nearly all cases terminate fatally. The perchloride of iron in full doses is the drug from which most may be expected; but its use may be combined with salicylates, or, perhaps, best with salol.

Chronic endocarditis is in children probably always a sequel of acute endocarditis. There is a sclerosis of the valve, leading to thickening, and, owing to the contraction of the fibroid tissue, to puckering, which renders the valve insufficient. In many, perhaps in the majority, of cases even in children, the fibroid thickening and consequent contraction involves the bases of the valves, and leads to narrowing of the orifice. The physical signs and consequences vary accordingly to the valve involved and the nature of the changes producing it.

The mitral is the valve most often diseased.

Mitral incompetence, due to endocarditis, may be produced by distortion of the valve and retraction of the chordæ tendineæ alone, but may be accompanied by narrowing of the orifice. A relative mitral incompetence without lesion of the valve may occur in fevers when the myocardium is weakened, in anæmia, or in dilatation from any cause. Permanent mitral incompetence throws increased work upon the left ventricle, and causes it to hypertrophy. Eventually it leads to dilatation of the pulmonary veins, arteries, and capillaries, leading to brown induration of the lung, and finally to hypertrophy of the right ventricle. When the compensatory hypertrophy of the ventricles is insufficient, or when from any cause it fails, the engorge-

ment of the pulmonary circulation is increased, and the growing embarrassment of the right heart leads to incompetence of the tricuspid valve, engorgement of the systemic veins, congestion, especially of the portal system, and finally to dropsy.

The **symptoms**, while compensation is maintained, are slight, but are usually more marked in children than adults. There is shortness of breath on slight exertion, the face is congested or slightly cyanotic, and the cutaneous venules are enlarged. The patients are liable to bronchial catarrh, and the expectoration is often blood-stained, or distinct hæmoptysis may occur. Complaint may be made of palpitation, or of uneasy sensations in the cardiac region, to be traced often to flatulence. The pulse is usually small and is often irregular, even with complete compensation. The *physical signs* vary with the degree of hypertrophy, with the length of time during which the condition has existed, and with the state of compensation. In children in whom the lesion has occurred at an early age, and has been followed by considerable hypertrophy, there may be very obvious bulging of the præcordia. The apex-beat is displaced outwards and downwards towards the axilla; it is strong and heaving if compensation is good, but weak, wavy, and diffuse if it have broken down. The area of cardiac dulness is enlarged, chiefly downwards and to the left. The first sound is more or less completely replaced at the apex by a murmur, which is usually blowing and is conducted into the axilla, and may be heard at the back near the angle of the scapula. Its intensity may be altered by a change in the position of the patient; thus it may be heard in the erect but not in the recumbent position, or *vice versâ*. The second sound is accentuated in the pulmonary region (the second interspace or third cartilage). When compensation gives way the action of the heart is weak and irregular, and the patient complains bitterly of being conscious of the heart's action, or of actual palpitation. Dyspnœa on exertion becomes more severe, and finally is never absent, the patient being unable to lie down. The overfilling of the pulmonary vessels causes an œdematous condition of the lung, determining cough and watery expectoration, often blood-stained. The overfilling of the systemic veins produces a cyanotic tint of the surface and œdema, generally beginning in the feet and ankles. The congestion of the portal system determines enlargement of the liver, gastric irritation, which often produces distressing vomiting, and gastro-intestinal catarrh. The urine is scanty and often albuminous. When dilatation has occurred the bruit may be very much diminished in intensity, and a soft tricuspid murmur may be heard at the lower part of the sternum.

Mitral stenosis is more common in females than in males. In rare cases it is congenital; as a rule it is produced by rheumatic endocarditis in early life. It is usually accompanied by some incompe-

tence. The valves may be thickened and so generally adherent that only a button-hole orifice is left, or, without much thickening, the valves may become adherent forming a funnel-shaped orifice. In rare cases, which are probably congenital, the valves may be little deformed, but the orifice is narrowed. With the button-hole or funnel orifice the chordæ tendineæ are shortened and distorted, or the musculi papillares are inserted directly into the valve. With this condition there is much less hypertrophy of the left ventricle than in insufficiency. In consequence of the obstruction offered by the narrow orifice, the left auricle becomes dilated and hypertrophied, there is backing up in the pulmonary vessels, and eventually dilatation and hypertrophy of the right ventricle; finally when dilatation is in excess of hypertrophy incompetence of the tricuspid is produced, with the consequences already mentioned.

In young children the hypertrophy on the right side may produce prominence of the fifth and sixth left costal cartilages and the lower part of the sternum. The apex-beat is often difficult to localize, being in reality under the sternum and produced by the right ventricle. The most characteristic physical sign is a thrill felt in the fourth or fifth left space over a limited area and immediately preceding the impulse. The area of cardiac dulness may be little altered, or it may be possible to discover a slight increase to the right. When compensation is established, the pre-systolic bruit is heard, a short, rough sound running up to the first sound which is loud and thudding. The combination is difficult to describe, but is very characteristic and hardly to be mistaken. The pre-systolic bruit is heard over a limited area to the right of the apex, and is not conducted into the axilla. It may be preceded by a diastolic bruit which may occupy the whole of the interval, or may be confined to the first part of the interval, when it is sometimes spoken of as post-systolic. As a rule there is no systolic bruit, though in some cases there is a faint or even a loud systolic murmur. The second sound in the pulmonary area is accentuated. Later on insufficiency of the tricuspid valve may lead to the development of a soft systolic bruit to the right of the sternum. When compensation fails the pre-systolic thrill and murmur may disappear, though a faint diastolic or post-systolic murmur may remain. When compensation is perfect there are no symptoms, and the patient may even live an active life past middle age without being aware that the heart is diseased. There is, however, a great liability to recurrent attacks of endocarditis, and to embolism, which occurs more frequently in mitral stenosis than in any other form of heart disease. Obstruction at the mitral valve if developed in early childhood interferes with growth, and the patients are usually of small build, sometimes obviously stunted. When compensation breaks down, the same symptoms ensue as in the

venous obstruction produced in the later stages of mitral incompetence.

Primary affections of the **tricuspid valve** are extremely rare. They occur either as a consequence of fœtal endocarditis, or in the course of pyæmia produced by disease of the umbilicus. The occurrence of tricuspid regurgitation as a consequence of disease on the left side has already been mentioned. It leads to systolic regurgitation into the auricle with pulsation in the cervical veins if the regurgitation be considerable and the heart strong. The area of cardiac dulness is increased to the right of the sternum, and a systolic murmur may be produced which will be best heard towards the lower part of the sternum, often over a very limited area. Tricuspid stenosis is a not uncommon form of congenital heart disease, but may be acquired, and is then secondary to disease on the left side, usually mitral stenosis, of which it is a most serious complication. It produces a pre-systolic thrill and short, low murmur heard to the right of the sternum near the base of the xiphoid cartilage. Tricuspid stenosis produces a marked, sometimes a very extreme, degree of cyanosis.

Disease of the **aortic orifice** is rare in childhood. Cases of **aortic incompetence** occur occasionally with characteristic hypertrophy of the heart, but as a rule the condition is associated with mitral insufficiency. The great hypertrophy leads to bulging of the præcordia, the impulse is forcible and felt over a wide area, the apex-beat is displaced outwards and downwards, and the cardiac dulness is increased in the same direction. A soft, long diastolic bruit, produced at the aortic orifice, is heard loudest at the second right interspace, but is conducted down towards the xiphoid. It is usually preceded by a short, rough systolic murmur, conducted upwards. A systolic murmur is often heard at the apex. It is either due to mitral insufficiency, or is conducted, a point often very difficult to decide; in some instances matters are further complicated by the presence of a pre-systolic bruit, heard on the left side in the fourth or fifth space near the sternal edge; it is attributed by Flint to a relative narrowing of the mitral orifice produced by the fact that the valves, owing to the hypertrophy and dilatation, are unable to swing fully back against the wall. The water-hammer pulse is not usually well developed in children, and visible pulsation of the arteries is seldom to be observed. While compensation is maintained there may be no symptoms, when it breaks down there are attacks of dyspnœa, cough due to œdema of the lungs, and irregular fever from recurrent endocarditis, which indeed may be the cause of the rupture of compensation.

Aortic stenosis, which may be brought about by an actual narrowing of the orifice, or by the obstruction offered by hardening and distortion of the valves, is very rare. It is produced occasionally by

fœtal endocarditis. The concentric hypertrophy which it produces causes less enlargement of the area of cardiac dulness than the eccentric hypertrophy of aortic regurgitation. The apex is displaced downwards and outwards, a systolic thrill may be felt at the base, and a bruit may be heard which may be conducted into the great vessels, and to the left of the sternum; it must be distinguished from the hæmic murmurs heard in that situation. These are less intense and harsh; there is no thrill and no hypertrophy; the murmur of aortic stenosis is best heard in the second right interspace, near the sternum; the impulse is strong and sharp; and the pulse is firm, slow, and of good tension.

It is common to find evidence of lesion at more than one orifice. The most frequent *combination* is mitral disease with aortic insufficiency; next, but usually when compensation has given way, the mitral and tricuspid are diseased together.

The **prognosis** of chronic valvular disease of the heart depends primarily on the perfection of compensation, and, owing partly to the steadily increasing demands made by the natural growth of the body, the prognosis is worse in children than in adults. The prospect is aggravated by the great liability to recurrent attacks of endocarditis, with aggravation of the lesion, and consequent early failure of compensation. The prognosis is better in mitral insufficiency than in mitral obstruction, if the risk of embolism, which is greater in the latter, be left out of account. The immediate prognosis of aortic disease is better in children than in adults, since the vessels are not atheromatous. On the whole, the prognosis of mitral disease, by far the most common in childhood, is not good if distinct hypertrophy occur before puberty, and is extremely bad if signs of failure of compensation appear before this age. Rest, careful dieting, and nursing may procure temporary amelioration, but a fresh breakdown usually follows a resumption of active life, and attacks of recurrent endocarditis are frequent. The risk of malignant endocarditis must also be taken into account. After puberty, if the patient be then in good health, and if compensation be perfect, the prospect is much brighter, and many such patients are able to live an active life, even to an advanced age.

Meddlesome **treatment** of chronic valvular disease of the heart is to be condemned. If there are no symptoms, no special treatment is called for. The patient should be placed under as good conditions as possible for the maintenance of the general health and of nutrition. It may be well to advise against occupations or games involving violent exertion, such as football or racing, whether on foot or on cycles, since it is known that the sudden strain on the heart thus caused favors, if it may not, indeed, determine fresh endocarditis, the great danger which attends chronic disease. When compensation

fails, rest, mainly in the recumbent attitude, is the first necessity, and the bowels should be freely moved by sulphate of magnesia, or some other laxative which produces copious watery evacuations. Sudden or extreme failure, with cyanosis and orthopnœa, may be met by cupping or, in extreme cases, by venesection. Digitalis is specially useful in dropsy, and it will often relieve this condition, if due to mitral incompetence, without rendering the pulse regular. For a child of ten ℥ viij of the tincture should be given three times a day, and increased daily until twice this quantity is taken. The urine should be measured, since the first indication that the dose of digitalis has become too high is afforded by a sudden decrease in the quantity passed; the digitalis should then be stopped for four or five days, or a week. At the commencement of the treatment a laxative dose of calomel should be given, and may be repeated after three or four days. In those cases in which digitalis fails to make the pulse regular, strophanthus sometimes succeeds. It should be remembered that dyspnœa may be due to hydrothorax, and will be relieved on aspiration of the fluid. When other means fail to remove anasarca, the patient may be placed in a semi-recumbent position, with the lower limbs dependent. This is often followed by great relief to the breathing, owing, apparently, to the draining of the fluid downwards. It will, however, then usually become necessary to remove the fluid from the lower limbs either by scarification or by the use of a capillary tube. In either case strict antiseptic precautions should be followed, and after twenty-four or thirty-six hours the patient should be put back to bed and the wounds induced, if possible, to heal. Palpitation, which is often a distressing symptom even before other symptoms of failure of compensation appear, is often produced by flatulent distension of the stomach or colon, accompanied by constipation, and is then relieved by a purgative; indeed, if the habitual use of laxatives is to be excused under any circumstances, it is in this condition. For the insomnia of failing compensation probably the best hypnotic is morphia in small doses, which also will be found to relieve the dyspnœa in many cases, and to quiet and strengthen the heart.

CHAPTER XXIV.

DISEASES OF THE MOUTH.

The Mouth—Dentition—Disorders of Dentition—Stomatitis—Partial Desquamation—Catarrhal Stomatitis—Membranous Stomatitis—Ulcerative Stomatitis—Aphthous Stomatitis—Thrush—Noma.

THE healthy infant breathes always through the nose, and the mouth is a potential cavity only; the tongue, when at rest, is in contact with the palate and with the cheeks and gums. The buccal secretions are scanty for the first two months of life, and the **saliva** has little action on starch; in infants suffering from atrophy and diarrhœa the salivary glands may fail to secrete any amylolytic ferment even at a later age. When dentition begins the saliva becomes copious, and its diastatic action on starch marked. Owing to the increased secretion and imperfect adaptation of the lips much saliva often dribbles away.

In the cheek, outside the buccinator and masseter, and lying upon both these muscles, is a lenticular mass of fat, about 1½ inches in diameter. Its function appears to be to prevent the falling in of the cheeks in the act of sucking, and the two bodies are commonly called the **sucking-pads**. In greatly emaciated infants these pads are not very noticeable, but above the age of six or seven months they do not waste with the rest of the body, and by their persistence give the child's face a characteristic and striking appearance. There are certain parts of the mucous membrane of the mouth which are especially vulnerable in infants. In most newly-born infants (52 per cent.) there may be observed one, or as many as five small, round, yellowish bodies in the mucous membrane of the palate, generally near the middle line. They are termed **epithelial pearls**, and consist of epithelial cells packed closely together. They are produced apparently by invagination during the process of closure of the palate. They have no pathological significance, and disappear, as a rule, during the second month of life at latest. If roughly handled during the process of removing scraps of curd from the mouth with the handkerchief, they may be injured and become the starting-point of ulceration.

There are two other points in the mouth of the infant which are specially vulnerable. The one is at and immediately behind the posterior edge of the hard palate, on either side; the other a little be-

hind the alveolar process. If the mouth of a young infant be held wide open two pale lines will be seen running up into the soft palate from the posterior end of the alveolar process of each upper jaw. In sucking, the tongue perhaps presses back on these two pairs of points, but they are more probably injured during the process of cleaning the mouth, when the lower jaw is depressed and the mucous membrane put on the stretch. Symmetrical shallow ulcers may thus be produced over these parts, and such ulcers are known by the name of **Bednar's aphthæ.**

Dentition.—As has been well said,[1] "Dentition is a continuous physiological process commencing in early fœtal life and terminating with the appearance of the wisdom teeth at the age of from eighteen to twenty-two, or even twenty-five years; but, whilst dentition may be said to be continuous, the eruption of the teeth is an intermittent process, the teeth appearing in groups and at certain intervals of time."

The eruption of the milk, or temporary, teeth begins at the sixth or seventh month and ends about the third year. The eruption of the permanent teeth begins in the sixth year and ends with the cutting of the third molar, at some period between eighteen and twenty-four years as a rule.

The *first dentition* is divisible into five periods:—

(1) *Sixth to Eighth month*	Two lower central incisors.
(2) *Ninth to Tenth month*	Two upper central incisors.
	Two upper lateral incisors.
(3) *Twelfth to Fourteenth month*.....	Two upper anterior molars.
	Two lower lateral incisors.
	Two lower anterior molars.
(4) *Sixteenth to Twenty-second month*.....	Four canines.
(5) *Eighteenth to Thirty-sixth month*.....	Four posterior molars.

This is the normal sequence, but variations are common. The upper central incisors may appear before the lower; the canines may appear earlier or later than usual, and similar irregularities may be observed in other teeth. Occasionally dentition begins very early, and children have even been born with teeth. Delayed eruption is far more common owing, probably, to the frequency of rickets.

The **second dentition** begins at about the end of the sixth year with the eruption of the first molar behind the second temporary molar; in the eighth year the central incisors; in the ninth the lateral incisors appear; and in the tenth and eleventh years the bicuspids replace the two temporary incisors. The permanent canines are cut about the twelfth year; the second molars about the thirteenth or fourteenth; and the last molars (wisdom) in early adult life (eighteen to twenty-four, or later).

[1] By Ballantyne, "Introduction to the Diseases of Infancy."

The **eruption of the teeth** is a physiological process, and may be attended by no local or general signs of disturbed health, but, like other physiological processes, it may be disordered and give rise to symptoms of irritation, both local and general. While the severity of this disorder of function and the frequency with which it occurs have been much exaggerated in the past, the opposite error must also be guarded against. In the adult the cutting of the wisdom teeth may be attended by a feeling of general illness, indisposition to make any exertion, drowsiness, headache, and slight elevation of temperature. Salivation is a frequent, if not invariable, accompaniment of the eruption of at least the earliest teeth, and some tenderness and itching frequently attends the distension of the gums. The child seeks to relieve the discomfort by chewing some hard substances, or the mother or nurse scrubs the gums with her finger; either process may result in producing stomatitis. In the eruption of the molars, the cusps may not come through simultaneously, and ulceration is very apt to take place, even in children in apparent health, under the flap of mucous membrane remaining over that of the crown. Once started this ulceration may spread by continuity along the groove between the teeth already cut and the gums. In infants who have cut only the two lower central incisors, a small ulcer not infrequently forms under the tongue, apparently from the pressure of the teeth. It heals, as a rule, in a week or ten days, either spontaneously or under simple antiseptic treatment.

Almost every disease to which infancy is liable has been set down at one time or another to dentition, but especially convulsions, diarrhœa, and various skin eruptions. It is possible that, in a child already predisposed to convulsions, the irritation attending disturbed dentition may turn the scale. Further, the thirst often present may induce the child to drink indigestible quantities of milk, and thus gastro-intestinal disturbance may be set up, and may cause diarrhœa or urticaria, or both. Beyond this it is difficult, with any confidence, to trace a connection between teething and the disorders mentioned. At the time of the second dentition, a good deal of discomfort may be, and commonly is, produced. The teeth about to be shed become loose, and in the act of mastication may easily inflict on the gum to which they are still attached an injury which will afford a point of attack to the pyogenic microbes so commonly present in the mouth.

The disorders of dentition seldom call for any active **treatment.** The routine custom of lancing the gums whenever they are found swollen and tender in an infant or child who presents no matter what nervous or other general symptoms is to be condemned strongly. In properly chosen cases, however, it may give instant relief. When a molar is almost through, and especially when the mucous membrane over it is anæmic or shows anæmic spots, the sur-

rounding mucous membrane being of a dark crimson, and when the child is restless, constantly champing its jaws together, a crucial incision may be made, and will sometimes give much relief, and procure a good night's sleep. In a few cases the thinned and anæmic mucous membrane over the crown is distended with fluid, and an incision into this removes the discomfort under which the child labors. Occasionally, when the lower incisors are being cut, the gums become very tender, and the child is very restless, champing its jaws, and tearing at anything it can put in its mouth; when with these symptoms, the finger can feel the edge of the tooth through the gum, when the mucous membrane at the summit is anæmic, and when, in particular, the child becomes quiet, and even holds out its mouth when the gum is touched, it is well to cut through the anæmic line, or to scratch it through with the finger-nail, previously rendered aseptic. The change in the child's attitude and aspect following this is often remarkable.

Incision, except under the circumstances mentioned, gives little or no relief, and may possibly derange or interrupt the natural process. If at an earlier stage of eruption the gums be swollen and tender, if the child be restless and irritable, and if, as sometimes happens, the temperature[1] runs up in the forenoon without other discoverable cause, it is well to give a dose of castor-oil and a mixture containing potassium citrate (2 grains every three hours at a year old) or bromide (3 grains every two or four hours to three doses). Rubbing the gums gently with the finger moistened with fresh lemon juice, with pure glycerine, or borax glycerine (made without water) gives temporary relief. Vigier recommends painting the gums with a solution of cocaine (1 per cent., see Appendix).

[Sodii Salicyl.,	gr. v
Cocain. Hydrochlor.,	gr. viij
Aq.,	℥j.]

The temporary teeth ought to be loosened and exfoliated by a process of absorption of the fangs without caries; but caries of the molars and incisors is extremely common, and in rickets the incisors and canines frequently undergo a process of disintegration at an early age, and are broken away down to the gums, where they show only as brown stumps. Sometimes the molars suffer from the same process, which is apparently not ordinary caries, and does not give rise to any obvious symptoms.

Ordinary caries at a later age, especially when it affects the molars, may be a cause of ulcerative stomatitis, of adenitis, and of general ill-health, owing to the absorption of septic material. The temporary teeth are often very much neglected, and it is surprising

[1] Eustace Smith, "Disease in Children."

how many young children, even of the wealthier classes, are permitted to evade the duty of brushing the teeth. If the child's health is fairly good, and if the carious teeth are not causing pain, they should not be extracted, as any teeth are, for the purpose of mastication, better than none. Children have been known to suffer from indigestion after extraction of stumps who had no such symptoms before. Still, such cases should be watched, having regard more especially to the risk of adenitis.

Stomatitis.

The mucous membrane of the mouth is more liable to become inflamed in infancy and childhood than after the age of puberty; the inflammatory process has a greater tendency to involve large areas, and its effects are often serious, owing to the risk of secondary infections, and to the fact that the tenderness may cause the child to refuse to take food.

Partial Desquamation.—In children under three years of age it is not uncommon to find that the tongue presents a peculiar form of irregular desquamation, which from the striking resemblance to a map, has been called the *geographical tongue*. Areas, which may be extensive, are red, and appear to be denuded of epithelium. Their margins are defined by curving edges of epithelium, which is a little paler than natural. The lesion appears to begin at several points by the swelling up of the epithelium, which becomes detached. The bare patch then formed extends in all directions, and by the coalescence of various areas the irregular patches are formed. The desquamation is very superficial, the deeper layers remaining. The cause of this curious condition is unknown; by some it is supposed to be due to the same agent as produces seborrhœic eczema, by others, including Unna, it is set down as a trophoneurosis. It is important to recognize that it is not, as supposed by Parrot, a sign of congenital syphilis. Children in whom geographical tongue is seen are usually suffering from some form of chronic gastro-intestinal disturbance, and the desquamation of the tongue ceases when the condition of the digestive organs is improved.

Catarrhal Stomatitis.—Catarrh of the mucous membrane of the mouth is the almost invariable accompaniment of certain acute specific fevers (*e. g.*, scarlet fever, measles) and of coryza. It is frequently associated also with dentition, appearing shortly before the eruption of each tooth or set of teeth; it may be caused also by cleaning the mouth with handkerchiefs, or by the use of dirty or old and cracked bottle teats. Catarrhal inflammation, limited in extent, especially over the hard palate, appears often to be determined by the retention of decomposing scraps of curd, especially in feeble infants. After the deciduous teeth have been cut they may early be-

come carious and determine stomatitis, acting either mechanically, by the irritation of sharp edges, or as a source of infection, owing to the decomposition taking place in the carious cavities.

The mouth feels hot and sticky. If the child is at the breast the mother may notice that the lips are burning, and that the child stops sucking frequently to cry. The mucous membrane will be seen to be swollen and œdematous. Redder patches may be seen here and there, and when this aspect is well marked the term *erythematous stomatitis* is sometimes applied. Or the mucous membrane may be generally somewhat pale from the œdema, and, if teeth be present, marked by depressions corresponding to them. There is some fulness under the jaw, due to adenitis, and some œdema about the glands. The breath is slightly offensive, and the temperature may be raised a degree or two.

Acute attacks, associated with the specific fevers or with dentition, commonly subside at once with the disease or as the tooth comes through; but when the cause persists, the condition may become subacute or chronic, or pass on to ulceration.

In the **treatment** of catarrhal stomatitis it is well to bear in mind that the catarrh may be kept up, if not, indeed, caused by the food and drink being given too hot. To clean out the mouth of an infant with a handkerchief over the finger is meddlesome. In feeble infants or young children who owing to pharyngeal or nasal obstruction breathe habitually through the nose, it may, however, be necessary to clean the mouth at night or after each meal. For this purpose a large soft camel-hair brush dipped in glycerine (or borax-glycerine) and water, equal parts, is the best means to employ until the child can be taught to wash out its mouth with boiled water which has become lukewarm, or a weak (1 per cent.) solution of borax. The mouth should always be cared for during specific fevers, especially measles and scarlatina, by the use of mouth washes (see Appendix) in children old enough to use them, or by gently painting the mucous membrane with borax-glycerine (two parts) and water (one part).

[(The following local applications for the mouth may be used :)

(a) Pot. Permang.,	gr. ij–iv	(b) Cupr. Sulph.,	gr. xxiv
Aq.,	℥j	Aq.,	℥j
(c) Resorcin.,	gr. iv–viij		
Aq.,	℥j		

(The following may be used as mouth-washes :)

(a) Thymol.,	gr. vj	Thymol.,	gr. iij
Boracis,	℥ss	Sod. Benzoat.,	℥iv
Spir. Rect.,	℥ij	Tr. Eucalypt.,	℥ij
Aq. Dist.,	ad Oj	Aq. Dist.,	ad Oj
		Borac Acid Cream.	Appendix.]

For marked catarrhal stomatitis no remedy is better than potassium chlorate, either in solution (1 to 2 per cent.), or in the form of lozenges or tabloids. In infants glycerine of boric acid or potassium chlorate may be applied with a brush. Whatever remedy is used, it must be used very frequently, and if lozenges or tabloids are preferred they should be broken into four or five pieces to be sucked separately.

Membranous Stomatitis (*aphthous s.*).—Catarrhal stomatitis when limited in extent, but acute in degree, may produce so much heaping up of epithelium and œdematous swelling of the superficial parts over limited areas as to give rise to patches resembling false membrane. These patches, which are of a yellowish, or greenish, color, and vary in size from a pin's head to a pea, may be seen on any part of the mucous membrane of the mouth or tongue; the plaques are surrounded by a zone of erythema which may be no more than a narrow band, or may extend over wide areas.

The **symptoms** are heat and soreness of the mouth, salivation, fœtor of the breath, and thirst, fever, restlessness, loss of appetite, swelling of the glands below the jaw, and œdema of the connective tissue about them. The condition is probably due to the local development of pyogenic organisms (the organism most commonly found is *staphylococcus pyogenes aureus*). It tends, as a rule, to spontaneous recovery, a process which may be hastened by the use of antiseptic mouth washes or creams. In weakly children, or in the subjects of gastro-enteritis, measles, scarlet fever, whooping-cough, pneumonia, and other acute febrile diseases, the inflammatory process may extend more deeply, and the patches then easily bleed. Eventually the plaques, which when hæmorrhage has occurred are much thickened by blood-clot, become loosened by ulceration. Their final detachment by this process is commonly attended by much pain and some hæmorrhage.

The **treatment** of the early stage of this condition consists in the use of general mouth washes (boric acid, potassium chlorate or permanganate), and the local application of a solution of perchloride of mercury (1 to 2 in 1,000), or of sodium salicylate and cocaine (see Appendix). In the later stages creams and other greasy applications, or glycerine of borax, or carbolic acid, or resorcin may be used with advantage, especially if crusts form.

Ulcerative Stomatitis.—The main etiological factor in the production of ulcerative stomatitis is traumatism, but if the mucous membrane is already in a catarrhal condition, ulceration may ensue upon injuries which in a healthy mouth would have no such effect. A carious, or sharp-edged tooth which has perhaps long existed, may then determine ulceration. In children who have cut only the two central incisors in the upper (or lower) jaw, an ulcer may thus be

produced in the mucous membrane at the point where the teeth impinge when the jaws are closed. Ulceration may occur at any part of the buccal or lingual mucous membrane, but there are certain sites where it is most frequently produced, or, at least, most frequently calls for treatment—at the edge of the tongue, or on the cheeks opposite the crowns of the teeth, in the groove where the gums overlap the teeth, and in the sulcus between the upper, but especially the lower lip, and the jaw, where the mucous membrane is reflected from the one to the other. Or the ulceration may begin in numerous scattered points sometimes grouped like herpes (herpetic stomatitis); this form, in fact, occurs frequently, but not always, in association with herpes labialis, or impetigo about the mouth. In the earliest stage a group of scattered spots are seen on the cheek or lip, or the side of the tongue; they are whitish, and slightly raised, consisting apparently of necrosed and swollen epithelium; this is quickly detached, leaving a shallow ulcer with sharp, or in some cases, undermined edges. These ulcers may quickly heal, or they may extend, and by confluence form ulcers of various forms, but commonly elongated.

In some cases ulceration of the gums extends to the bone and causes extensive necrosis; in association with acute infectious diseases, especially scarlet fever and typhoid fever, extensive sloughs, which may have a gangrenous character, occasionally form and involve parts of the jaw, the tonsils (leading perhaps to ulceration into the internal jugular vein), or soft palate, leaving after recovery a perforation.

The **treatment** of ulcerative must be governed by the same general considerations as that of catarrhal stomatitis. In the early stage potassium chlorate is a valuable remedy, but is less effectual later. The main difficulty in treating ulceration of the mouth, when it has become thoroughly established, is to ensure that the antiseptic used reaches all parts of the ulcer, as is well illustrated by the extreme obstinacy of ulceration in the groove between the gums and teeth. Moreover the copious salivation which often accompanies the stomatitis tends to wash away any local application. Sulphur ointment (ung. sulph. (B. P.), adeps lanæ, vaselinæ āā) meets the indications, and is an effectual but disagreeable remedy; a lanoline cream may be used as a basis for various antiseptics, potassium chlorate, borax, resorcin, etc. (see Appendix), and has the advantage that the lanoline appears to penetrate well, and remain for some time attached to the ulcerated surface. Painting the ulcer with a solution of mercury perchloride (1 to 2 per 1,000) in the early stage, or with a solution of sulphate of copper (gr. xx to ℥j) or nitrate of silver (gr. v–x to ℥j), or touching the small accessible ulcers with lunar caustic two or three times a week, helps to bring about a healthier condition. A solution of

potassium permanganate (1 per cent.) is well spoken of, and a solution of sodium salicylate and cocaine is also recommended (see Appendix). In any case it should be borne in mind that ulceration of any part of the mucous membrane of the mouth which is much used, as, for instance, in the sulcus between the lower lip and the jaw, may cause great pain, and so render the child restless, irritable, and averse to food. Under such circumstances small doses of opium, which have the further effect of diminishing the excessive flow of saliva, may be of great service.

Aphthous stomatitis is an infectious disorder derived from cattle suffering from aphthous fever (foot and mouth disease). In children infection takes place through the ingestion of the milk of an infected cow, as a rule, but it may also be transferred from one child to another residing in the same house and using the same drinking vessels.

The earliest **symptoms** are fever accompanied by salivation, indisposition to take the bottle, and often by diarrhœa. Red spots then appear on the tip or sides of the tongue, or on the lips or palate. At the centre of the red spots vesicles form, and persist, surrounded by a red zone, for two or three days. The vesicle then bursts, leaving a sharp-edged shallow ulcer covered with a puriform false membrane. As a rule, only some eight or ten vesicles form and the ulcers do not coalesce. In such cases the disease is mild and tends spontaneously to recovery, though a phlyctenular eruption, probably due to infection from the mouth, may appear on the cheek, chin, arms, or hands. While the ulcers persist, the mouth is sore and painful, the fever continues, and the sub-maxillary glands may become enlarged. When the vesicles are numerous the resulting ulcers may coalesce and involve not only the palate, but the tongue, lips, and even the pharynx. All the symptoms are then much aggravated and the child may pass into a typhoid condition. After recovery from a mild attack, a second, and even a third may occur if the use of the infected milk be persisted in.

The **prognosis** is generally good.

The **diagnosis** can only be made with certainty in those cases in which the vesicles are observed.

Much difference of opinion exists as to the frequency with which this disease occurs in the human species; infants under two years of age are certainly more liable to it than older children.

When there is reason to suspect that stomatitis is of this nature, it is, of course, desirable to change the source of milk supply, or, if this be not possible, to boil the milk. The mouths of other children who have been taking the milk should be carefully examined, and simple antiseptic mouth washes prescribed. In the **treatment** of the disease a strong solution of sodium salicylate (20 per cent.) has been

strongly recommended as a mouth wash ; when there is much pain a solution of cocaine hydrochlorate (2 per cent. for infants, 5 per cent. or 10 per cent. for older children) should be applied to the parts with a brush. In order to prevent intestinal complications it is advisable to give salol (gr. ij to iij four times a day to an infant ; gr. v to a child of two to three years), or naphthaline, or salicylate of naphthol.

Thrush is a term often loosely applied to any soreness of the mouth ; it is here limited to the special affection of the mouth produced by the *saccharomyces albicans.*

This form of stomatitis, which is seen in its characteristic form in marasmic infants, especially in those suffering from gastro-intestinal derangements, presents three stages. In the first, or erythematous stage, the mucous membrane is of a dusky red, the tongue is dry and glazed, its papillæ enlarged, and the secretions in the mouth are acid. The infant sucks with some difficulty, but does not appear to be in pain, and there is no obvious glandular enlargement. After a day or two the creamy membranous growth characteristic of the second stage begins. Small points and patches, at first of the most brilliant opaque white, form on the dorsum, tip, and sides of the tongue, often also on the cheeks and lips. Spreading rapidly, these patches cover the parts affected with a white layer, which on the tongue especially is particularly uniform, as though a finely granular white paint had been spread over it with a spatula. On the cheeks and hard palate it is, if present, usually less uniform, and has rounded scalloped edges. The creamy layer can be removed, commonly with great ease, and the mucous membrane is then seen to be reddened, but not ulcerated. The membrane forms again quickly after removal. In the third stage the membrane loses its brilliant white color and uniformity ; it turns yellow or gray, and becomes detached in places, or if the patient is sinking, it assumes a dark gray or brown color and dries into cakes.

Though usually limited to the mouth the membranes may extend beyond the fauces to the pharynx, œsophagus, stomach, and even to the small and large intestine ; the infection may become established in the vagina, but not, it is said, in the rectum. In robust infants the infection does not spread so widely or rapidly, and is commonly limited to a few scattered patches on the tongue or hard palate. In them it produces few or no symptoms beyond some slight indisposition to suck freely. In the severer forms the child refuses to suck, and the movements of the tongue are slow and slight. Even in them, however, thrush is rather of importance clinically as an indication of an extreme state of prostration than in itself a cause of that condition.

The **pathology** of the condition has nevertheless given rise to much speculation. If a particle of the membrane be detached, cleansed with potash, and examined under the microscope it will be seen to

consist of epithelial cells, food débris, and of matted filaments with some rounded bodies resembling spores; leptothrix filaments and other micro-organisms may also be seen. The larger filaments belong to the specific organism, but it appears that they are not a true mycelium. Cultivated on solid media it forms white colonies, which are found to consist of rounded cells enclosed in a refracting capsule; these cells multiply by gemmation. In fluid media the cells become elongated, giving origin to a false mycelium. True spores form only in sugary media. Much importance has been attached to the acidity of the secretions of the mouth observed in thrush, but it appears that the saccharomyces will grow as well in neutral or alkaline as in acid media. It will not, however, grow in saliva, which agrees with the clinical observation that thrush occurs most often during the first two months of life, when the secretion of saliva is scanty or absent, or during febrile or wasting diseases which tend to suppress the secretion. It is probable that the sequence of events is that particles of curdled milk retained in the mouth during sleep and in the weakness of fever or marasmus undergo acid fermentation, determining an erythematous stomatitis, which then becomes the seat of the specific infection with saccharomyces. The mycelial-like elements of the saccharomyces grow between the epithelial cells, which undergo necrosis, and may even force their way into the submucous tissue or between the muscle fibres; in rare instances they find entrance into the lymphatics, and have been found in internal organs (kidneys, spleen, brain). The origin of the infection cannot always be traced, but the disorder has occurred in epidemics in lying-in and foundling institutions, and under such circumstances is probably conveyed from one infant to another by the india-rubber teats of the feeding bottles, or by other utensils used in common.

Thrush is not in itself the cause of much discomfort or danger; it may be *treated* by glycerine of borax or glycerine of mercury perchloride applied with a stick tipped with cotton-wool, or by solutions of potassium permanganate (1-per-cent.) or borax (2- to 4-per-cent.) applied in spray. At the same time it is usually desirable to give salol or other intestinal antiseptics, or to treat any gastro-intestinal disorder which may be present. If the white layer reaches far back and invades the pharynx, Baginsky recommends the administration every two hours of a drachm of a solution of resorcin (gr. ij–iv in ℥j).

Noma is a peculiar form of spreading phlegmonous inflammation leading rapidly to gangrene; it attacks the cheek (cancrum oris) and vulva.

It is now a rare disease. Its victims are children between the ages of two and seven or eight years who have been brought into a cachectic condition by a recent attack of measles, scarlet fever, or

some other acute infectious fever, by malaria, or more rarely by chronic gastro-enteritis or an insufficient diet.

In cancrum oris the earliest symptom is a thickening of the cheek between the skin and mucous membrane. Whether this is always preceded by ulceration of the buccal surface is doubtful, but as a rule at least ulceration is present when the case is first seen. The induration spreads rapidly, and accompanying œdema renders the whole cheek brawny ; the skin becomes involved in the inflammatory process, and is generally red, with a purplish tinge. On the buccal surface gangrene may occur early and spread rapidly both towards the surface, causing sloughing of the skin, and internally to the gums, the periosteum, and the bones. The deformities resulting may be extensive.

The local process does not produce much pain. It is accompanied by general symptoms of a low adynamic type. There is not much fever and the temperature may not be at all elevated. Septic diarrhœa, and septic broncho-pneumonia are apt to occur, rendering the prognosis exceedingly grave. Pulmonary gangrene and gangrene of the extremities have also been observed.

Noma of the vulva may occur as a complication of cancrum oris, or independently. It runs a rapid course, and may quickly determine extensive gangrene.

This form of spreading gangrene is due to a short bacillus which in cultivation forms, by juxtaposition, long threads. Occasionally several cases have occurred in succession in foundling institutions, but as a rule the mode of infection cannot be traced.

The **treatment** must consist in scraping the surface, followed by the free application of the acid solution of nitrate of mercury or of nitric acid and the subsequent use of antiseptic applications. The operation must be performed under an anæsthetic. The patient must be given a diet which is nutritious but light (predigested). Alcohol is often required in full doses.

CHAPTER XXV.

DISEASES OF THE UPPER RESPIRATORY PASSAGES.

Rhinitis—Acute Laryngitis—Chronic Laryngeal Catarrh—Papilloma of the Larynx—Acute Pharyngitis—Acute Tonsillitis—Otitis Media—Chronic Pharyngitis—Adenoid Vegetations—Chronic Tonsillitis—Deformities of the Chest produced by Naso-Pharyngeal Obstructions—Retro-Pharyngeal Abscess—Respiratory Spasm.

Rhinitis.—Infants sneeze a good deal during the first few days of life. There are no distinct signs of catarrh, and the sneezing is due to the mechanical irritation of dust in the air, but for the whole of the first year they are very liable to acute rhinitis, which is often complicated by catarrh of the naso-pharynx, pharynx, and mouth, and sometimes by bronchitis. After exposure to cold the infant begins to sneeze, to breathe through the mouth, owing to obstruction of the nasal passages caused by the hyperæmia which is the first stage of catarrh, and to have some difficulty in suckling. Shortly a thin mucous secretion begins to run from the nose, and excoriation of the upper lip is apt to ensue. There is some elevation of temperature, and the infant is restless and does not sleep well. The secretion becomes muco-purulent, and owing to its stickiness and to its drying in the nose and at the nostrils obstruction to respiration continues. The infant takes the nipple into its mouth, but is quickly compelled to relinquish it to take breath. When this has occurred twice or thrice it becomes restless, cries, and may refuse altogether to suck.

Gastric catarrh is a frequent complication, and rapid wasting may ensue owing to an insufficient quantity of milk being taken and imperfectly digested. Attacks of inspiratory dyspnœa may occur, which in rare cases have been traced to falling back of the tongue in inspiration. Relapses and repeated attacks are common. In young infants the **diagnosis** must be made from syphilitic rhinitis (*q. v.*) and from diphtheria. Much swelling of the nose, and the early appearance of a muco-purulent secretion, especially if it have an offensive smell and be associated with depression and somnolence, should excite suspicion of diphtheria, and the fauces and pharynx should be examined thoroughly. [The question can be settled only by bacteriological examination, which should always be made in suspicious cases. See chapter on Diphtheria.] At a somewhat later age the

probability that the rhinitis marks the commencement of measles or whooping-cough must be borne in mind.

The **treatment** should consist of the injection, very gently, of mild antiseptic lotions (boric acid) and the application of soft ointment (*e. g.*, boric acid) to the anterior part of the nasal cavity with a camel-hair brush ; or the ointment may be applied on plugs of cotton-wool, large enough to distend the nostrils slightly, which are left in place for half an hour. If the secretion is muco-purulent and irritating, a mild white precipitate ointment is to be preferred. Should the infant be unable to suck it must be fed with the spoon.

Acute laryngitis varies very much in severity. In severer cases there is in addition to catarrhal inflammation much sub-mucous œdema, and it may be inflammation, which may extend to the cartilages. In this severe form it is not a common affection except as a complication of diphtheria, sometimes of scarlet fever or measles, more rarely still of typhoid fever, or small-pox. The symptoms are identical with those produced by laryngeal diphtheria, and it is often impossible to be certain that the laryngitis is not diphtherial. The reader is, therefore, referred to the article on diphtheria.

In the milder form to which the term *laryngeal catarrh* is often applied, there is a catarrh of the mucous membrane which is red, swollen, and at first dry, but afterwards covered with a watery mucous secretion. The catarrh may begin in the subglottic part of the larynx, and may be attended by some sub-mucous œdema. As a rule it is secondary to acute catarrh of the nose and pharynx (coryza) or of the bronchi, and in some is apparently produced by direct extension of the inflammatory process. One attack predisposes to another, and a slight exposure to cold, or to dusty air, may determine an attack in children who have once suffered. This liability is one of the most frequent sequelæ of measles.

The **symptoms** are in the main the same as those of sub-acute laryngitis in the adult, with one characteristic addition—"croup"—which is due to a reflex spasm of the glottis-closers. An infant, or young child, who has, perhaps, begun to suffer from an attack of coryza during the day, awakes suddenly struggling for breath ; after, perhaps, a few husky coughs the chest becomes fixed in expiration, the face congested, the eyes suffused, and the attack terminates by a long, noisy, high-pitched inspiration. The child then begins to cry and cough, and both cry and cough may be a little husky, or quite natural. Such an attack causes great alarm in a household,[1] but by the time the child can be seen it is probably sleeping peacefully. It is, however, very likely to awake again once or twice in the same night with similar attacks. The next day it appears quite

[1] See Henoch's graphic description (*Vorlesungen*, S. 332 ; *New Syd. Soc. Trans.*, vol. i., p. 357).

well, or it may present the ordinary symptoms of slight coryza, or the voice may be a little hoarse, and the cough "croupy"[1]—that is to say, loud and clanging. Attacks of croup may recur during the following night, but this is not the rule, as they seem to be associated usually with the dry, early stage of laryngeal catarrh and to cease when secretion becomes established.

Treatment during or immediately after an attack of croup should be directed to relieving the laryngeal congestion by hot compresses, or poultices, or a mustard leaf to the front of the neck, and the mitigation of the dryness of the mucous membrane by keeping the air of the room moist and warm (65° F.). In children old enough to use it an inhalation of steam with tincture of benzoin gives relief. The routine use of emetics is not to be recommended, but after the croup has passed away ipecacuanha in small doses repeated every hour or every two hours is of use in promoting secretion. It is not desirable to add opiates in the early stage, but later, if the cough be frequent, the compound tincture of camphor or a linctus containing a small dose of morphine, or codeine (gr. $\frac{1}{20}$ to gr. $\frac{1}{6}$) may be given in the evening. When a child has once had an attack of croup the prophylactic treatment is of much importance. It should be warmly clad, taken out in the open air as much as possible, but not in very damp weather; if old enough gymnastic exercises are useful, and in summer a bracing climate, and an out-of-door life.

Chronic Laryngeal Catarrh occurs in children as a sequel to the acute affection, and occasionally in association with chronic pharyngitis and adenoid vegetations. Inherited syphilis appears to be the cause in some cases in young children, and in older children chronic laryngitis is sometimes associated with sub-acute relapsing rheumatism. The mucous membrane is thickened and there is hoarseness, and chronic cough. Occasionally the thickening is progressive and the obstruction to respiration may necessitate tracheotomy.

Symptoms resembling those of chronic laryngitis are occasionally found to be due to new growths in the larynx, of which the commonest is **papilloma**. In such cases there are usually many grayish white warty growths on and about the cords. In other cases the growths are larger, more vascular, and even pedunculated. When this is the case sudden fatal asphyxia may be produced by impaction of a growth between the vocal cords. The earliest age at which papillomata have been observed is fourteen months.[2] Medicinal treatment is useless. Partial extirpation is apt to be followed by recurrence, and thorough extirpation necessitates thyrotomy, which

[1] "Croup" is the loud inspiration due to glottic spasm; "croupy cough" the short loud clanging or ringing cough of slight laryngeal catarrh.

[2] In a child who had been hoarse since six weeks old (Bornemann, *Jchr. f. Kinderhlkde,* Bd. xxxv., S. 333).

may leave permanent hoarseness. If sources of irritation—the passage of air in breathing, but especially the violent movements of coughing—are removed, the growths tend to undergo spontaneous atrophy. The best treatment[1] appears to be to perform tracheotomy, and to let the child wear a soft rubber tracheotomy tube until all trace of obstruction has ceased and the voice has regained its natural quality. It may be necessary to continue the use of the tube for several years.[2]

Acute inflammatory affections of the **pharynx** and of the adenoid tissue in relation with it, are of great importance in childhood owing to the frequency with which they occur, and the difficulties in diagnosis which they often present. As a rule the mucous membrane and the adenoid tissue (tonsils and pharyngeal tonsils) are involved simultaneously, but the one or other tissue may be the more severely affected.

Although acute pharyngitis is usually determined by infection or exposure to cold, or by one of the specific diseases—scarlet fever, measles, small-pox, chicken-pox, erysipelas, acquired syphilis—family predisposition, insanitary dwellings, and chronic catarrh must be reckoned as predisposing causes.

The symptoms of **acute pharyngitis** are commonly less severe than in adults; there is some elevation of temperature, which may reach 101°–102°F. for a few hours, but soon falls. It is accompanied by some increase in the pulse and respiration; the breathing is a little noisy; there is some cough, and the child hawks up a good deal of glairy mucus. Pain on swallowing is less than in adults, but complaint may be made of pain about the angle of the jaw, and the glands there will be found enlarged and tender. On inspecting the throat in the early stage red patches are seen, a little later a uniform redness and swelling of the mucous membrane, which is covered by a thin, frothy mucus. The mucous glands are usually swollen, and rounded prominences about the size of a pin's head form, thickly set, which may break down, causing small superficial erosions; the pillars of the fauces may be of a purplish color and œdematous. Some enlargement of the tonsils is frequent, but in a first attack it is usually slight. In seven cases a thin whitish false membrane may form, generally on the tonsils or the pillars of the fauces.

In other cases, usually attended by extensive œdematous inflammatory swelling of the mucous membrane of the fauces and pharynx, the **tonsils** are more acutely inflamed. They are large, bright red, soft and tender. Some cases, to which the term *follicular tonsillitis* is applied, show groups of yellowish points due to superficial suppuration beneath the epithelium. These may break down, leaving shallow ulcers.

[1] Hunter Mackenzie, *Brit. Med. Journ.*, 1896, vol. ii., p. 609.
[2] Railton, *Ibid.*, 1898, vol. i., p. 488.

In other cases again, which are undoubtedly infectious, the inflammation of the tonsils is more deeply seated; they are swollen, firm, and the mouths of the lacunæ are plugged with a cheesy purulent secretion (*angina lacunaris*). There may be a thin white false membrane on the tonsils, and, as the cases occur with special frequency during times of diphtheria prevalence, the diagnosis is often difficult. The disorder lasts two or three days, and ends often by crisis. In these forms of acute tonsillitis both organs are usually affected and commonly to about the same degree.

Finally, the inflammation of the substance of the tonsils may be sufficiently acute and extensive to produce *suppuration*. The swelling of the tonsils is very great, so that they meet in the middle line, or if one be affected more than the other, as is often the case, it bulges across the middle line, until it is in contact with its fellow on the other side. There is a great deal of mucous secretion, which often dribbles from the mouth, much pain on swallowing, and enlargement of the glands at the angle of the jaw (often mistaken for the tonsils). This *acute phlegmonous tonsillitis* may end in suppuration (*a*) in the tonsil, (*b*) more often in the connective tissue about it (peritonsillar abscess), or (*c*) in the lymphatic glands, in which case the abscess presents externally behind the jaw.

Inflammation of the tonsils and pharynx may be so intense that *gangrene* ensues, causing great destruction of tissue, which has led, in some cases, to opening up of the carotid, and profuse hæmorrhage. This severe form has been observed chiefly as a complication of scarlet fever. When the tonsils are the part mainly affected, the symptoms are more severe and acute, and the fever, which is often preceded by shivering, a rigor, or even by convulsions, is high, 103°–104° F. Severe tonsillitis is rare in infants, and comparatively uncommon in young children; at about six or seven years of age it begins to be more frequent, and it is very common about the age of puberty.

Since acute pharyngitis and tonsillitis may be the earliest manifestations of several acute specific diseases the **diagnosis** is often exceedingly difficult. Inquiry should be made as to the probability of recent exposure to infection, by scarlet fever and diphtheria in particular, and as to the acute specific diseases from which the patient has suffered previously. Tonsillitis and acute pharyngitis also frequently prevail epidemically in schools. The only safe rule is to regard every sore throat as possibly infectious, and to isolate the patient from other children. Very sudden onset, with vomiting or convulsions and a high temperature, is in favor of scarlet fever; "strawberry" tongue will excite suspicion, and the rash will declare itself within twenty-four hours. If due to measles the pharyngitis will probably be accompanied by coryza, bronchitis, and congestion of the face. Acute pharyngitis, accompanied by paroxysms of cough,

especially if these end in vomiting, is, in children who have not suffered from the disease, due probably to whooping-cough. At an early stage it may be quite impossible to exclude diphtheria; it must be remembered that a sore throat, especially in children who have already suffered from diphtheria, may be diphtherial though no membrane be present throughout the case. Bacteriological examination will afford confirmatory evidence, but too much reliance must not be placed on a negative result. A thin white membrane, especially if semi-transparent and associated with tonsillitis, does not justify the immediate diagnosis of diphtheria. The child should be isolated and watched; in such cases the membrane often clears away very rapidly and the bacteriological examination is negative.

Otitis is a frequent and most serious complication of pharyngitis. It is particularly apt to occur as a complication of the acute specific diseases, especially of scarlet fever and measles, but it is not infrequent as a complication of simple pharyngitis and of acute tonsillitis. It is due to infection of the middle ear by extension from the air passages, but its occurrence is favored by the obstruction of the Eustachian tube produced by swelling of the pharyngeal mucous membrane. The inflammation is at first catarrhal and is soon attended by suppuration. Subsequently the inflammatory process may extend to the mastoid cells, to the cranial bones, the meninges, and the brain (*vide* "Abscess of the Brain"). The microbes found are the *diplococcus pneumonicæ* of Fraenkel, the *bacillus pneumonicæ* of Friedländer, the *streptococcus pyogenes*, and the *staphylococci* (*albus* and *aureus*). Of these Fraenkel's *diplococcus*, and the *streptococcus*, which is the microbe most often met with in the otitis of scarlet fever,[1] are those found most frequently.

The **symptoms** of otitis media are acute pain in the ear and the side of the head, tenderness over the ear and behind the angle of the jaw, with usually some redness and swelling of the external canal; if the drum can be seen it will be found red or bulged forward. If the child be already suffering from an acute fever the onset of the otitis is very apt to be overlooked. It should be suspected if the child show that it is in constant pain by whining continuously, with an occasional sharp cry. The diagnosis will be confirmed if the child pick or rub at its ear, and if the intelligence be dulled, or if the temperature remain high, after, in the ordinary course of the disease, it should fall, and especially if it assume the suppurative type. An infant suffering from acute otitis media cries unceasingly, and utters occasionally shrill, quavering screams. It bores its head into the pillow or holds it motionless on the nurse's shoulder, and very often tears at the external ear. It is feverish, restless, and refuses food. In other cases the symptoms closely resemble those of posterior basal

[1] Blaxall, *Brit. Med. Journ.*, 1894, vol. ii., p. 116.

meningitis, retraction of the head being marked, vomiting and convulsions not infrequent, while symptoms directing attention to the ear are absent. Sooner or later in most cases the drum is perforated, a thick purulent discharge escapes, and all the symptoms are at once alleviated. The occurrence of mastoiditis will be indicated by tenderness and inflammatory œdema over the mastoid process. It is, however, more often a complication of chronic than of acute otitis media.

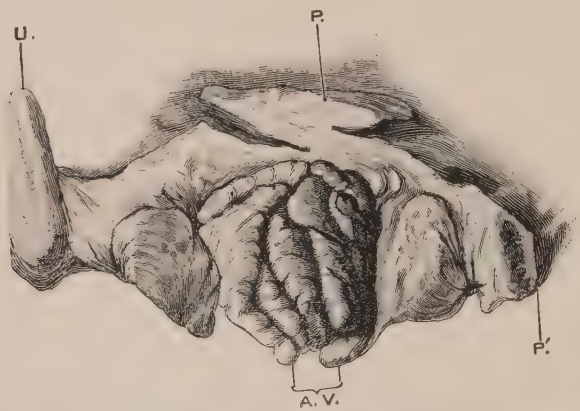
In the **treatment** of acute pharyngeal catarrh, fomentations or a cold compress to the neck, and warm washes (boric acid) for the mouth and as gargles, give relief. The fauces and pharynx should be brushed lightly three or four times a day with glycerine of tannin or borax, or strong solution of perchloride of iron and glycerine (equal parts) or chloride of zinc. Small doses of potassium chlorate in decoction of cinchona, with perhaps some ipecacuanha are useful, but antipyretics are seldom called for. In follicular tonsillitis the internal administration of sodium salicylate relieves the pain and apparently hastens resolution. Quinine is also recommended. Guaiacum in small doses, if given at the onset of an attack of tonsillitis, will sometimes cut it short. A lozenge containing gr. ij–iij, should be sucked every two or three hours. As local applications, the glycerine of tannin and of perchloride of iron are of value. Sucking small pieces of ice relieves the pain, and the local application of a solution of cocaine (5 per cent.) diminishes dysphagia. A brisk purge at the onset of symptoms is to be recommended.

In acute pharyngeal catarrh, whether primary or secondary, the liability to the occurrence of acute otitis media must always be kept in mind; much may be done for its prevention by the use of antiseptic mouth washes, and by the application of warm solutions of boric acid by a coarse spray or syringe through the nose, followed by the application of an antiseptic ointment with a camel-hair brush. Pain in the ear may be relieved by hot fomentations, or a leech to the mastoid, and by the instillation of a small quantity of a watery solution of cocaine and atropine (of each 2 per cent.). If these measures fail to give relief in a short time the tympanic membrane should be punctured by a vertical incision in the posterior section. Owing to the smallness of the parts, and the very oblique position of the tympanic membrane in relation to the external auditory canal in infants, it is often difficult to obtain a satisfactory view of the drum, but if the symptoms point to ear disease, and in cases in which they resemble those of posterior basal meningitis, it is advisable at once to perform paracentesis of the tympanum. The puncture heals easily, so that if the operation be skilfully performed no harm, even if no good, results. Field states that in some of the cases in which paracentesis fails to give relief this is because Politzer's bag has not

been used to dislodge the accumulated pus. After the acute symptoms have ceased attention must still be given to the naso-pharynx, which commonly remains in a condition of chronic inflammation with lymphatic hypertrophy; in children deafness may require the use of Politzer's bag at intervals for many months. Chronic discharge from the ear calls for treatment by warm mild antiseptic lotions carefully used, and the insufflation of boric acid in fine powder or of a powder consisting of equal parts of sodium chloride, bicarbonate, and biborate. During and after the acute attack enlargement of the lymphatic glands behind the angle of the jaw may call for attention; of various local applications which may be used in the acute stage, probably belladonna fomentations are the most effective in preventing suppuration.

Chronic pharyngitis in children is usually a sequel of repeated attacks of acute catarrh of the pharynx and naso-pharynx. The

FIG. 48.



Adenoid vegetations of the naso-pharynx (drawn, from a post-mortem specimen prepared by Dr. A. T. Rake, by Miss M. Gordon, L.R.C.P. and S. Ed.). U, uvula. P, P', cut edges of soft palate. A, V, adenoid vegetations.

commonest form is granular pharyngitis. The earliest age at which I have seen this condition was in an infant aged four months. It becomes very common about two years of age, and after that age some 70 per cent. of London children of the poorer classes have granules in the pharynx. The granules are produced by swelling of the mucous glands with infiltration of the surrounding sub-mucous tissue. The intervening mucous membrane may be healthy, inflamed, or atrophied. The number of granules varies greatly, from two or three to twenty or more. They may be scattered irregularly or gathered into two bands which occupy the salpingo-pharyngeal fold (pharyngitis lateralis); in this situation the granules are partly hid-

den by the pillars of the fauces, or by the tonsils if enlarged, but come into view when the child swallows. The average size is about that of a split pea. If scattered, and few in number, they produce no symptoms, but if numerous they cause a sensation as of a foreign body in the throat, and constant hawking and coughing. These symptoms are more marked in lateral pharyngitis. They are much aggravated and may be accompanied by some pain on swallowing during the attacks of sub-acute catarrh to which the mucous membrane is in these cases greatly predisposed. Cough may be very severe, and mucous expectoration in older children copious. Probably two-thirds of the children supposed by their friends to be the subjects of chronic bronchitis are, in reality, suffering from granular pharyngitis. The condition is often associated with adenoid vegetations of the naso-pharynx, but by itself does not produce any obstruction to respiration. After a time the repeated attacks of catarrh may lead to some sclerosis and atrophy of the intervening mucous membrane.

When the granules are scattered and few in number they do not call for any treatment. They shrink gradually, become flattened, and finally disappear. When numerous the discomfort in swallowing, the cough, and the frequent attacks of catarrh, render interference desirable. The only effectual remedy is to destroy the centre of each granule with a fine galvanocautery, if that be available. If it be not, a fairly satisfactory substitute is to touch each granule with London paste applied with a glass rod. It is a useful precaution to paint the throat with cocaine solution if the granules to be attacked are numerous or situated on the lateral folds. As a rule not more than half a dozen granules should be dealt with at one sitting.

The adenoid tissue of the upper part and roof of the naso-pharynx is liable to hyperplasia, and chronic inflammatory changes analogous to those which produce granules in the pharynx. The overgrowth throws the mucous membrane into folds, as is well shown in the preceding illustration (Fig. 48). This condition, to which the term **adenoid vegetations** is applied, may be present very early in life and may even, it is said, be congenital. The size of the nasal passages and of the naso-pharynx, however, varies very greatly in different individuals. When they are narrow, and when, as often happens in these cases, the palatine arch is high and narrow, and the anterior nares small or collapsed, an amount of adenoid overgrowth, relatively small, may block the air passage. An instance of this condition is afforded by the girl aged twelve, photographs of whom are reproduced in Figs. 49 and 50. She presents the "adenoid facies," but the nasal obstruction is due in great measure, if not entirely, to the extreme narrowness of the nasal passages.

The typical expression, the attitude, and some of the deformities

of the chest produced by adenoid disease are well shown in Fig. 51, from a photograph, for which I am indebted to Dr. StClair Thomson. The face wears a dull, heavy expression, the mouth is constantly open showing crowded irregular teeth, which, together with the gums, are in many cases dry and coated. The upper lip is caught up, the lower droops. The nose is pinched but clumsy, the nostrils narrow, owing to paresis of the alæ nasi. The child is often deaf, owing to blocking of the pharyngeal orifice of the Eustachian tube. For the same reason there is a great liability, owing to retention of secretions, to otitis media. A transverse vein at the root of the nose is commonly enlarged.

Fig. 49.

Fig. 50.

Nasal obstruction associated with congenital narrowness of the nasal passages.

The adenoid overgrowth is almost invariably attended by catarrh and a muco-purulent, sometimes bloody, secretion, which may be seen on the posterior wall of the pharynx, and may escape from the nostrils during sleep, staining the pillow.

The symptoms are inability to breathe through the nose, chronic "cold in the head," noisy respiration by day, and snoring during sleep. The voice has a peculiar nasal, toneless, or "dead" character, and pronunciation is defective; there is difficulty especially in pronouncing the nasal consonants *n* and *m*.

The reflex nervous symptoms attributed to adenoid vegetations are legion, and their enumeration would form a Rabelaisian catalogue of little practical value, since the evidence upon which the supposed connection is founded is commonly very insufficient. Two only

FIG. 51.

A boy, aged 4¾, suffering from adenoid vegetations of the naso-pharynx, showing the characteristic expression, attitude, and deformities of the chest. (From a photograph in the possession of Dr. StClair Thomson.)

seem worthy of mention: (1) The condition of mental dulness and inability to fix the attention, to which Guye has given the name, aprosexia. It may be compared with the "stupid feeling" produced by acute coryza, and may very quickly disappear after operation for the removal of the vegetations. (2) Nocturnal enuresis, which may

certainly be aggravated if not produced by adenoid vegetations. The connection is probably to be found in the light, broken sleep from which these children suffer.

[A certain number of these cases, as well as some cases of enlarged tonsils (see below), are of a tubercular nature, and such are undoubtedly the starting point of chronic tubercular processes, and are apt to be associated, as an etiological factor, with enlarged cervical glands.]

The **symptoms** having suggested the presence of adenoid vegetations, the **diagnosis** may be confirmed, or perhaps negatived, by inspection or by introducing the finger behind the soft palate, with the pulp forward. The convoluted folds of mucous membrane will then, if present, be felt, "like a bag of worms."

If the overgrowth has attained dimensions sufficient to produce definite symptoms, especially any notable interference with respiration, the only effectual **treatment** is to scrape away the redundant folds. This operation should not be postponed in the hope that the child will "grow out of" the adenoids. It is true that the obstruction to respiration becomes less as the naso-pharynx becomes more roomy, but this process is retarded by the loss of function due to adenoid vegetations. Moreover, the continuance of the obstruction during the early years of growth of the chest may entail irremediable deficiency in its development and permanent deformity (Fig. 51). There is a difference of opinion as to whether the child should be given an anæsthetic for this operation, but in my experience as much, or rather, as little, good can be done by scraping away at the time of examination, as much as the finger-nail can detach, as by any more elaborate operation without an anæsthetic. In well-marked cases such imperfect operations are not to be recommended, since they are almost invariably followed by recurrence. Cure depends on the production of a sufficient area of raw surface to ensure a good deal of cicatricial retraction. The main risks of the operation are the entry of blood or detached vegetations into the air passages (which may be minimized by keeping the head dependent) and septic basal meningitis. The operation should, therefore, not be undertaken until the general health has been got into as good a condition as possible, and until the local condition has been improved by astringent and antiseptic applications where this is found possible, though the small size of the naso-pharynx often renders their application very difficult. After the scraping, antiseptic douches (boric acid) should be used.

Chronic tonsillitis is the term commonly applied to a condition of enlargement of the tonsils, due to overgrowth of the lymphoid tissue with attendant fibroid hyperplasia. It is secondary to repeated attacks of pharyngeal catarrh or tonsillitis, and is in some cases as-

sociated with, if it be not directly due to, the presence of tubercle bacilli. [On the other hand, many of these cases are but manifestations of rheumatism, and, as mentioned above (see chapter on Rheumatism), necessitate careful watching for other manifestations of this diathesis, especially endo-pericarditis.] The enlargement may be so great that the tonsils meet in the middle line. The lymphatic glands near the angle of the jaw are enlarged secondarily in most cases, and the distortion of the neck thus produced may be considerable. The tonsils are at first soft, but in time may become exceedingly hard. The surface may be smooth, or coarsely granular. The mucous membrane may appear healthy, or it may be in a condition of chronic catarrh, with much mucous secretion, and accumulation in the lacunæ of offensive cheezy matter which may eventually become calcified (tonsillar calculus).

The **symptoms** are a sensation of a foreign body in the throat, chronic cough, nasal voice, breathing through the mouth, and snoring during sleep. The breath is often foul, owing to decomposition of the cheesy masses in the crypts. The patients are very liable to catarrh of the pharynx, with consequent increased swelling of the tonsils, aggravation of all the symptoms, and free expectoration.

As in the case of adenoid vegetations, all kinds of reflex nervous symptoms have been attributed to chronic enlargement of the tonsils. The most important secondary effect, however, is the deformity of the chest described below, to the production of which tonsillar hypertrophy appears to contribute.

The only effectual **treatment** is tonsillotomy, that is to say, the removal of the most prominent part of the enlarged gland. It is followed by cicatricial contraction. Local applications may subdue intercurrent catarrh, but have not the least effect on large hard tonsils. If operation is refused they may be recommended as prophylactics against catarrh and consequent increased fresh enlargement of the tonsils. Tonics, iron, and cod-liver oil, and change of air may be advised, and if the nutrition is well maintained, and the child gets plenty of exercise in the open air, the tonsils eventually shrink, and as the naso-pharynx enlarges, cease to obstruct respiration. The danger is, however, that the development of the chest, and of the frame generally, may receive a check from which it never recovers.

Deformities of the chest produced by *naso-pharyngeal obstructions*.—The obstruction to respiration produced by overgrowth of the adenoid tissues of the naso-pharynx leads in a large number of cases to deformities of the chest walls. These changes are particularly marked in children who suffer also from rickets. The commonest deformity is (1) pigeon-breast. The lower and lateral parts of the chest are retracted during inspiration by the diaphragm, so that a horizontal groove is produced, while the sternum is thrust

forward, and often bent at the junction of the manubrium with the body. A modification of this is (2) the so-called shoemaker's or funnel chest, in which the angle in the sternum is in the middle of the body, the lower part of the bone being pulled back, so that in some cases a deep hollow is formed. If the obstruction begin when the chest walls are more firmly ossified, the result is to produce (3) a long narrow flat chest (Fig. 51), the transverse diameter of the lower part is diminished, while the upper front is flattened, or even hollowed, a condition which undoubtedly predisposes to phthisis. In robust children, in whom the obstruction occurs after the age of eight or nine years, no deformity of the chest may ensue. If, however, the obstruction is accompanied by attacks of asthma the lungs become emphysematous, and (4) the barrel chest is produced.

Retro-pharyngeal abscess is not a common affection in infants, but it occurs in them more frequently than in children. Two classes may be distinguished: (1) the so-called idiopathic, which may be a sequel of measles or scarlet fever, or of stomatitis, tonsillitis, or disease of the naso-pharynx, though more often no cause can be assigned; and (2) tuberculous abscesses, secondary to vertebral caries. The suppuration occurs in the connective tissue outside the pharynx.

The early symptoms—restlessness, an expression of pain during suckling and its sudden cessation followed by crying, are not characteristic. After a time, perhaps a week or ten days, the breathing becomes snoring, especially during sleep, which is much disturbed, suckling is more difficult and more evidently painful, and milk is returned through the nose. The infant breathes through the nose, and respiration is labored, so that the symptoms resemble those of laryngitis; but the voice is usually unaltered. In some cases there is torticollis. Examination of the pharynx shows at first only general redness and catarrh. Dyspnœa and distress may be severe, and there may be some cyanosis of the face and distension of the jugular veins before a localized swelling can be seen on the front of the vertebral column, usually in the middle line. A confident diagnosis can only be made when a soft swelling can be felt with the finger in the pharynx. It is round or oval, and conveys to the finger the sense of fluid contents. If left to itself the abscess may burst into the pharynx, and flood the larynx, causing sudden death. Early incision is therefore imperative, and gives immediate relief. The left forefinger placed against the lower edge of the swelling should be used as a guide, and the incision made with a tenotomy knife. As soon as the incision, which should be half an inch long, has been made the infant should be turned on its face, so that the pus may flow out of the mouth. Only in rare cases has the abscess been known to fill again, but cases of burrowing septic abscess occur. As a rule, the diagnosis, when once the abscess has attained sufficient size to be recognized by the

sight or finger, is easy, but when, as happens in rare cases, it is situated low down, it becomes a matter of great difficulty, since it is almost impossible to get a view of the parts, and the diagnosis must be made by touch alone. An abscess due to caries is a more chronic affection, may present in the neck, and can best be treated by external incision.

Respiratory Spasm.

Children are liable to certain disturbances of the nervous mechanism of respiration, which produce symptoms of an alarming, and sometimes even of a dangerous, character.

There is a form of infantile respiratory spasm, however, to which the term **congenital laryngeal stridor** is applied, though it is not always truly congenital, which does not endanger life nor interfere with growth.[1] At birth, or within the first fortnight of life, the inspiration is noticed to be noisy, hoarse, or croaking, ending sometimes in a short crow; expiration is silent, but may be grunting. There is recession of the epigastrium and the lower part of the chest, the alæ nasi may move, but there is no cyanosis and no distress. The stridor is not constant, it varies in degree, may disappear altogether for a time, or may be interrupted from time to time by full, long, noiseless inspirations. Excitement increases the stridor, but it disappears or becomes much less marked when the child cries. Sleep diminishes and, when sound, stops the stridor. The infant can breathe through the nose, and sucks without difficulty. The stridor is probably due to some defect in the higher nervous centres, producing incoördination, or spasm of the laryngeal muscles. In some cases there is an undue backward curvature of the epiglottis, which has been considered by some to be a cause, by others a consequence, of the stridor. The stridor may occur quite independently of laryngeal catarrh. It is not related to rickets, and cannot be connected with tetany with any confidence, though carpo-pedal contractions occur in some cases when the stridor is greatest.

Respiratory spasm in children is a paroxysmal affection nearly always associated with rickets, often with tetany, of which it may be the earliest symptom. The attacks sometimes end in, or alternate with, convulsions.

Beyond the almost invariable association with rickets[2] nothing can be said definitely as to its etiology. In some cases there is laryngeal spasm only, but in the more severe forms there is complete temporary arrest of all the movements of respiration, due apparently to an inhibition of the respiratory centre.

[1] Dr. John Thomson has given an excellent description of the condition in the *Edinburgh Medical Journal* (Sept., 1892).

[2] Bull found in 100 cases of spasm, unmistakable signs of rickets in 94, and more doubtful indications in 3 others. *Jahrb. f. Kinderhlkde,* Bd. xxxvii., S. 401.

Very frequently the attack comes on when the child wakes, and *night terrors* are often due to this cause. It may be provoked by crying, as is the paroxysm of whooping-cough, by coughing, by hiccups, by any alarm, or by exposure to a cold draught of air. The paroxysms may occur as often as twenty times in the twenty-four hours. During attacks of inspiratory stridor the sterno-mastoid and other accessory muscles of respiration are in violent action, and there is recession of the epigastrium and the lower parts of the chest. Emphysema of the upper and anterior parts of the lung, and collapse of the lower may thus be produced, and favor the occurrence of broncho-pneumonia to which such children, owing to their rickety condition, are already predisposed. A severe attack produces much exhaustion and the frequent repetition of such attacks endangers life. The nature of the paroxysms varies in different cases. The commonest form is a slow prolonged expiration followed by a normal inspiration. A slight attack, such as this, causes the child little distress, and may even pass unperceived by the friends. The next most frequent form is the converse of this—that is to say, a normal expiration is followed by a prolonged inspiration. The inspiration may be attended by spasm of the glottis-closers, and when this is sufficiently severe it is accompanied by a long, high-pitched, crowing note. The term **laryngismus stridulus** is applied to the forms in which this occurs. In some cases both expiration and inspiration are prolonged. None of these forms are immediately dangerous, though the loud inspiratory cry is alarming. More alarming, and more immediately dangerous, since sudden death has often occurred, are cases in which expiration is followed by a prolonged pause during which the chest is motionless, and the face grows rapidly cyanosed. This may end in a free inspiration with or without the crowing sound (with which all immediate danger ceases), or by very imperfect attempts, short or long at inspiration, during which there is no sound, the chest walls recede and the muscles of the mouth and the alæ nasi work.

The **prognosis** depends upon the frequency and severity of the paroxysms. It is not good when they are numerous and severe, owing to the exhaustion which they produce and the liability to intercurrent affections of the lungs. The paroxysms, at first slight, may become more serious, and the possibility of sudden death from asphyxia and of the occurrence of general convulsions should not be hidden from the parents.

The **diagnosis** is usually easy if the child can be seen in one of the attacks. The absence of laryngeal catarrh and coryza, the more chronic course, and in many cases the expiratory character of the spasm, will serve to distinguish respiratory spasm from the laryngeal spasm of croup. Whooping-cough, owing to the history of infection, the regular stages by which it develops, and the special characters

of the cough which precedes the inspiratory spasm can hardly be mistaken for the respiratory spasm here described.

Treatment during the attack cannot be very effectual. The clothes should be loosened, cold water sprinkled on the face and chest, and the skin chafed with the hand. If a hot bath is available the child should be put into it, and a douche of cold water poured over its chest. Between the attacks sedatives, such as potassium bromide or chloral, are of little use, though when the child is having attacks very frequently they may be of some temporary advantage. Henoch prefers opium or morphine (gr. $\frac{1}{6}$ or more according to age), the hydrochlorate or acetate of morphine. The main indication is to treat the underlying rickets (*q. v.*).

CHAPTER XXVI.

ACUTE BRONCHITIS, BRONCHO-PNEUMONIA, AND PNEUMONIA.

Acute Bronchitis and Broncho-pneumonia: Pathology; Symptoms; Prognosis; Treatment—Acute Lobar Pneumonia: Etiology; Pathology; Symptoms; Complications; Diagnosis; Treatment.

Acute bronchitis and broncho-pneumonia, which are among the most serious and common diseases of childhood, are very closely related to each other clinically and pathologically. Both occur with great frequency as complications of measles, whooping-cough, diphtheria, and other acute infectious diseases. In many cases, in which they are apparently primary, they are preceded by coryza, pharyngitis, or tracheitis. The extension of the inflammatory process is associated with the spread of one or more micro-organisms. In bronchitis the microbes found most commonly are the *staphylococcus pyogenes* and the *streptococcus pyogenes;* in broncho-pneumonia the *pneumococcus* of Fraenkel, and the *pneumobacillus* of Friedländer. But the microbes which most often cause bronchitis may, under circumstances favorable to their entry into the alveoli, produce pneumonia, and the *pneumococcus* and the *pneumobacillus* may cause only bronchitis. Children attacked by bronchitis have often suffered for some time from gastro-intestinal disturbance with diarrhœa of offensive mucous stools. The diminished power of resistance due to the deterioration of general health produced by gastro-intestinal disease no doubt favors the development of bronchitis or broncho-pneumonia, and in other circumstances it is not to be assumed that either is always, or indeed commonly, to be traced to infection from a previous case. The bronchial or pulmonary tissues damaged by debilitating disease, by the inhalation of irritants, or by the changes induced by exposure to cold, easily become infected. One or more of the varieties of the microbes commonly associated with acute bronchitis or broncho-pneumonia are present in the mouth in a large proportion of all cases examined. Further, these microbes are to be found in overcrowded rooms, and may be conveyed from one person to another by drinking vessels used in common, especially in hospitals and asylums. Thus is to be explained the extreme frequency and severity of broncho-pneumonia among children suffering from measles and whooping-

cough, nursed in overcrowded tenements or in institutions in which special precautions are not taken to avoid infection.

Pathology.—Acute bronchitis may affect any part of the bronchial tree, and is more serious the finer the bronchi involved. To the most severe cases, in which the finest bronchi are involved, the term capillary bronchitis has been applied, but it is probable that in all these cases the alveoli are also attacked, and that we have to do with broncho-pneumonia added to the bronchitis. In broncho-pneumonia there is a general inflammation of all the tissues of the lung—the bronchi, pulmonary alveoli, and lymphatic system, and if the part affected be near the surface the pleura also may be involved. At the same time there is more or less inflammation of the large bronchi, and of the medium bronchi in many other areas than those in which consolidation occurs. In fact, bronchitis, congestion of the pulmonary tissue, and areas of consolidation are present together, but in varying degree and extent in different cases and in different parts of the lungs in the same case. Thus the whole or greater part of a lobe may be consolidated, while in other parts of the same lung and in the other lung we find bronchitis with, as a rule, disseminated patches of lobular pneumonia. Some enlargement of the bronchial lymphatic glands necessarily attends bronchitis and broncho-pneumonia, and chronic adenitis may remain as a sequel. The presence of these enlarged glands appears to favor recurrence of bronchitis. In many cases these glands become tuberculous, and in some at least the tuberculous adenitis is the primary lesion to which recurrent attacks of bronchitis or broncho-pneumonia are secondary (see p. 169).

In *acute bronchitis* there is at first hyperæmia, and serous infiltration of the bronchial mucous membrane, which becomes swollen but remains dry. Upon this condition ensues diapedesis, with accumulation of leucocytes beneath and between the epithelial cells, detachment in greater or less numbers of ciliated cells, increase in the number of mucous cells, swelling of the mucous glands and copious secretion from these sources. The surface is thus rendered moist, at first by a tenacious mucous, and later by muco-purulent, material. When the inflammation is persistent it may involve eventually the bronchial muscles and the elastic tissue, and thus determine more or less extensive and permanent dilatation of the bronchi. In slight cases the trachea and large bronchi only are involved; in more severe, the medium bronchi, also; and the most severe the smallest bronchi, producing "capillary bronchitis," which, on account of the rapidity with which severe symptoms develop, is sometimes called "suffocative." The infective process extends thence to the pulmonary alveoli, and it is probable that capillary bronchitis is always accompanied by some alveolar catarrh, which, if

death does not occur at an early date, passes on quickly to distinct *broncho-pneumonia*. The inflammation may extend not only, as above described, by contiguity, but also by inspiration of infective secretions. During the deep inspirations which precede and follow cough infective muco-purulent matter in the larger bronchi may be sucked down into the smallest, and there start inflammation of the lobule. Such a plug of mucus may act as a valve, permitting some air to escape during expiration, but preventing entrance during inspiration. In this way all the air may be expelled, and the lobule collapse. Apart from any valvular action, if a plug occlude a bronchiole, the air is then absorbed from the lobule which therefore collapses. This condition of **atelectasis** is an important factor in broncho-pneumonia, since the collapsed lobules easily become involved in the inflammation of neighboring lobules, or infected from the plug of mucus. Atelectasis is favored by any condition which renders full expansion of the lungs difficult—by the congestive thickening of the mucous membrane and the tenacious secretions produced by bronchitis, by the thoracic deformities of rickets, and by prolonged lying on the back in one attitude. It occurs most frequently at the borders of the lungs, especially the lower border, but often involves large areas in the posterior portion of the lower lobe. The collapsed area is sunk below the general surface of the lung, is of a dark red or purple color, and shows a uniform red surface on section. It sinks in water, but can be insufflated unless inflammation have already commenced.

Acute bronchitis varies much in severity. In a case of moderate severity the child, after perhaps suffering for a day or two from coryza, begins to have a dry cough, the breathing is a little hurried and labored, the pulse is quickened, and the temperature is raised, touching 100° or 101° F. at night. The child is restless and thirsty, but refuses food. The skin is moist and the face flushed. The chest expands well, and there is no dulness on percussion; on auscultation sibili are heard here and there, especially at the back, but are often masked by a loud rhonchus which has its point of maximum intensity over the large bronchi in the interscapular region. The sounds are inconstant, rhonchus may disappear after cough, and the points at which the sibili are heard may change in the course of a few minutes. As secretion from the mucous membrane begins the sibili give place to loose mucous râles, but as a rule children under five years do not expectorate. In a more severe case the inspiration is more hurried, the sibili of the early stage are heard in all parts of the chest, and are more constant, and the mucous râles of the later stage are smaller and more numerous. It is useful to bear in mind the dictum of Graves that the more numerous the sounds heard at any one point to which the stethoscope

is applied the smaller the bronchi involved. The pulse is rapid, 120–130, and the face may be pale, or even slightly cyanosed, and the lips bluish. The temperature reaches its highest daily point generally in the evening; it may be 102° or 103°, or only 100° F. or less. A low temperature is an unfavorable sign, generally observed in cachectic children. In these, and especially in rickety children, the expansion of the lower part of the chest may be defective, so that there is recession in the lower axillary regions, in the episternal notch, and in the epigastrium. The recession is greater when there is laryngitis, or obstruction of the trachea and larger bronchi by tenacious mucus. Much recession generally means some collapse, and with collapse we are on the verge of broncho-pneumonia.

The **prognosis**, both as to recovery and as to duration of symptoms, varies greatly. In a well-nourished child the symptoms may reach their maximum in a couple of days, and begin rapidly to subside in two or three days more, so that the patient is convalescent at the end of a week. In other cases, especially in cachectic children in whom the primary systemic reaction is not well marked, a condition of subacute broncho-pneumonia is very apt to supervene, and the case may drag on for weeks or months, or chronic bronchitis may become established.

It is often impossible to say when acute bronchitis becomes complicated with **broncho-pneumonia**. When the child has been suffering previously from no more than a subacute attack of bronchitis, the onset of the pneumonic complication is more easily distinguished. The child is noticed to be peevish and restless, changing its attitude at short intervals. The cheeks are flushed, the skin dry, and the rapidity of respiration is increased. There is a loose cough, and the child cries a good deal; it refuses food, but suffers much from thirst. At night all the symptoms are aggravated and the temperature rises to 102°, 103° F., or even higher. The alæ nasi move, and inspection of the chest shows that some of the accessory muscles of respiration are in action. At one or more points, most often near the angle of the scapula or at the base, sub-crepitant râles may be heard. These are often obscured by rhonchi and sibili in the larger bronchi. A little later the respiration over this area becomes bronchial; while sub-crepitant râles may be heard in other parts of the chest. The signs of consolidation are generally more pronounced on one side, but are commonly present on both. Vocal resonance is increased, and it is possible to detect some diminished resonance on percussion.

But the onset of broncho-pneumonia may be very much more acute. A child, after suffering for some days from an attack of bronchitis not presenting features of special severity, is seized suddenly with dyspnœa and a short painful cough. The face is pale,

with a dusky blueness about the nose and lips. The expression is anxious; the eyes prominent, and the nostrils dilate with each inspiration. The child, if old enough to sit up, leans upon its hands, and all the accessory muscles of respiration are called into action. All its energies are absorbed in attempting to get air into its lungs, and it does not interrupt these efforts to cry. From time to time the accumulation of mucus in the large bronchi and trachea renders the breathing more or less stertorous. Presently, by a short choking cough, the mucus is dislodged, and swallowed, and the child puckers its face as though about to cry, but seldom makes any sound. Respiration is very rapid, reaching perhaps 80 in the minute, and the pulse runs up to 140 or 160. The skin is dry and pungent. The child refuses food, takes little notice of its surroundings, and sleeps little, if at all. Physical examination of the chest commonly fails to reveal pulmonary changes sufficient to account for the violence of the symptoms. Sub-crepitant râles may be heard over large areas of the back and axillæ, but the percussion note is little if at all impaired. A condition of such extreme gravity and distress cannot long be endured. The circulation begins to fail, the face becomes gray and haggard, the eyes glassy, the skin cold and perspiring. The pulse grows quicker, 160 to 180, irregular, and often uncountable at the wrist. Respiration becomes more shallow and less and less effectual, while mucus accumulates in the larger bronchi. Finally, the child grows drowsy, and passes into a condition of somnolence which ends usually in death.

The **prognosis** of broncho-pneumonia depends in part on the extent of lung involved, in part on the general condition of the patient before the attack, or on the nature of the general disorder which it complicates, and in part on the patient's surroundings. In cases of the type last described the prognosis is very unfavorable, as the immediate danger to life is great, and even in those in which the symptoms are less severe, and the physical signs more limited, the course is very variable. In some cases the symptoms and physical signs clear away in a week or a fortnight. In others in which they continue with alternate remissions and exacerbations for a month or more the question whether the broncho-pneumonia be not really tuberculous will arise, but will often be difficult, in fact, impossible, of solution, unless the progress of the case can be watched for some time. In the more prolonged cases death is brought about rather through exhaustion than by the intensity or extent of the disease.

Treatment.—A child suffering from even slight *acute bronchitis* should be kept in a well-warmed room, which should have as little furniture as possible; the air should be kept free from dust, and, if necessary, moistened artificially. A laxative or purgative should be ordered, and a simple linctus containing ipecacuanha.

[Alkaline: Vini Ipecac., ♏iij–vj
Pot. Bicarb., gr. iij
Aq. Anethi, ℥j
Appendix.]

Hot fomentations, if skillfully applied at the onset and renewed two or three times, often give relief. Free perspiration may be provoked in robust children, who should, however, under these circumstances be kept strictly in bed. The child may be given warm drinks, such as hot milk diluted with water; or, in the houses of the poor, weak tea with milk, or the old-fashioned remedy—camomile tea. In any case the draught should be copious and hot. Spirit of nitrous ether, formerly a favorite domestic remedy, operates in a similar manner. Superior to all these is a hot bath for twenty minutes, starting at 95° F. and raised to 104° F. After it the patient should be dried quickly with a large rough towel, put into a warm bed, and wrapped up in a blanket, which may be withdrawn in an hour. Or the bath may be followed by a warm pack. The routine practice of giving expectorant drugs at all stages of bronchitis is not to be commended. In the earliest stage, when the chest is full of sibili, ipecacuanha will afford relief to the feeling of tightness; the best way to administer it is in small doses (for a child of one year, ♏iij–v) every two hours for ten or twelve doses. At a later stage, when numerous mucous râles are to be heard in all parts of the chest, ipecacuanha and other expectorants are useless or, rather, so far as they have any action, harmful. The difficulty at this stage is in the expulsion of the large amount of mucous secretion formed. Diffusible stimulants are open to the same objections; the best stimulant, when it becomes necessary to administer one, is alcohol—brandy, champagne, mulled claret, egg-nog, or the brandy mixture of the B. P. Opium and morphine should be used with great caution, but can not render important service in the treatment of bronchitis in the latter stages, when respiration is obstructed by mucous accumulations, and when the cough occurs in paroxysms or is so frequent as to prevent sleep. A convenient remedy is the compound camphor tincture, to the amount of about ♏xx in twenty-four hours, in six doses (for a child one year old), or the hydrochlorate of morphine in solution, or a single dose in powder in the evening.

[(The compound camphor tincture may be given as follows:)
T. Camph. Co., ♏ij–iv
Acid. Hydrochlor. Dil., ♏j
Vin. Ipecac., ♏iij–v
Glycerin., ♏x
Ad. Caru., ad ℥j
Appendix.]

In the early stage, in place of ipecacuanha, antimonial preparations may be used. Apomorphine also is highly recommended as an expectorant.

[Apomorphine Linctus,

Apomorphine Hydrochlor.,	gr. $\frac{1}{64}$
Acid. Hydrochlor. Dil.,	♏j
Syrup. Limon.,	℥j
Aq.,	ad ℥j

For children over 8 years. Appendix.]

When moist sounds in the chest show that there is much mucous secretion, children old enough to understand should be encouraged to cough; younger children should be induced to shift their position, or taken out of bed wrapped up in a blanket to promote coughing. Very young children do not expectorate, but the mucus expelled from the air passages is swallowed. Young and feeble children should not be entirely confined to bed, nor permitted to remain too long in the recumbent attitude. When the amount of secretion is very large an attempt may be made to induce vomiting should the general condition not forbid it. For this purpose a large dose of ipecacuanha (℥j of ipecacuanha wine, or ipecacuanha in powder with tartarated antimony) may be given. It is not always easy to induce vomiting, nor wise to repeat the dose. If vomiting does not ensue, the child should be taken up, the tongue depressed and the fauces tickled, in the hope that the mechanical stimulus may reinforce the action of the drug. Jürgenson recommends strongly the hypodermic injection of apomorphine.

Severe bronchitis, the so-called capillary bronchitis (which probably always means bronchitis associated with broncho-pneumonia), should be regarded as a very serious disease, calling for most careful treatment.

In *broncho-pneumonia* the fever rarely in itself constitutes a danger, and the use of antipyretic remedies is not to be recommended. All drugs belonging to this class, with the exception, perhaps, of quinine, have a depressing effect upon the heart, and the danger to life is in a breakdown of the heart and of the nervous forces. The liability to pulmonary collapse must be borne in mind, since it is both dangerous in itself and a precursor of extension of the pneumonia. Infants and young children should not be left for long hours motionless in bed. They do much better in a nurse's arms, since in practice this involves frequent shifting of position. A warm bath (80° to 85° F.) should be given for fifteen minutes; or a hot bath (95° to 97° F.) for a rather longer time. The good effect of the bath may be judged by the diminution in the respiratory rate. If the child be robust and not exhausted, from half a gallon to a gallon of water at a temperature of about 65° F. may be rapidly poured over the chest before the child is removed from the bath. This will induce deep respiration, followed, probably, by a fit of coughing. The bath may be repeated twice or thrice in the course of the twenty-four hours. The tempera-

ture of the bath and of the water used for the cool douche at its end may be varied according to the effect produced. If exhaustion already exists when treatment is commenced, or if the child be slightly cyanosed, the general cold douche should be replaced by a stream of cool water poured on the nape of the neck, about the vertebra prominens, from the spout of a kettle or from an india rubber siphon, for ten or twenty seconds. When removed from the bath the child should be rapidly dried with a rough towel, dressed in a flannel nightgown, and put into a warm bed.

The baths may be replaced by a wet pack, which is always to be preferred in weakly children. The temperature of the water used for making the pack and the extent of the body to be covered by it must be determined by the general condition of the child: and good results may be obtained in cases of moderate severity by the cold pack applied round the chest alone. In children of robust constitution, with high temperature and signs of extensive broncho-pneumonia, the pack may be wrung out of ice-cold water, or an ice poultice may be applied to the chest; but its effect must be carefully watched, and the method should not be used unless the patient is under frequent observation. Under similar circumstances, dry cupping over the part most affected is often followed by much improvement in the breathing. In the early stage, when the face is flushed and sibili predominate in the lungs, a steam-tent over the bed often gives relief; but it is not desirable to continue its use indefinitely.

Expectorant remedies also are generally beneficial in the early stage, especially ipecacuanha, given in the manner recommended above. The child should be carefully fed at regular intervals, the best food being milk diluted with seltzer water or barley water, and in older children egg beaten up with sherry or brandy and water (about half an egg to a dessertspoon of sherry or ℥xxx of brandy). Infants at the breast, owing to the dyspnœa, are commonly unable to obtain a sufficient quantity of milk, which must be drawn off and administered in a spoon. The great danger is heart failure, and against this, unfortunately, little can be done. Caffeine, either by the mouth or hypodermically, or subcutaneous injections of camphor, may be of service.

In children beyond five or six years old strong coffee, to which a small quantity of cognac is added, may be of service. When the child is tending to recover, the quantity of nourishment given should be increased, and it should, if possible, be moved by day into a second room, which has previously been well warmed and aired. It is advisable to begin the administration of cod-liver oil at as early a date as possible, and in children who are already habituated to this remedy, it may be given as soon as the temperature falls.

Acute lobar or fibrinous pneumonia (*pneumonic fever*)[1] is a specific inflammation of the lung due to infection by the pneumococcus. The lung is not the only organ which may be infected by this microbe; pericarditis, endocarditis, meningitis, laryngitis, and otitis may be produced by it, either independently or, more commonly, as complications of pneumonia. As already said the pneumococcus may produce either broncho-pneumonia or lobar pneumonia, but affection of the whole lobe of a lung, or more than one lobe, by the acute inflammatory process gives rise to a clinical type of disease very well characterized in adults, and to be recognized, though its symptoms are often less typical, in children.

Etiology.—The pneumococcus is present in the saliva of about one healthy person out of five, but in a much larger proportion, four in five, of those who have had pneumonia months or even years before the examination. The infection may be derived directly from a person suffering from pneumonia, and the disease may develop at once or after an indeterminate period, during which the pneumococcus remains in the mouth. The pneumococcus may retain its vitality for some time in articles of clothing or furniture soiled by expectoration; in this way may be explained the so-called "house pneumonia," one case occurring after another in the same house. "Family pneumonia" (the predisposition of particular families) may be accounted for in part by the survival of the pneumococcus in the mouth of a person who has once had the disease and its transference to other members of the family, where it remains until these individuals are eventually subjected to conditions which favor the fixation of the pneumococcus in the lungs.

Exposure to cold must be reckoned among the most important of the *determining causes* of cases of pneumonia not secondary to the acute infectious diseases. Secondary pneumonia occurs with greatest frequency after typhoid fever and influenza; but it is not infrequent in association with diphtheria, scarlet fever, and erysipelas, and is occasionally a complication of small-pox and of acute rheumatism and malaria. These diseases determine the onset of pneumonia in two ways—by diminishing the resistance of the body and increasing the virulence of the pneumococcus. Cold, which can be recognized distinctively as an antecedent in a quarter or a third of the cases, probably acts reflexly on a lobe or lung, preparing it to receive the pneumococcus. Symptoms may set in within two or three hours of the exposure. A blow on the chest is the determining cause of a small proportion of the cases of pneumonia in the adult, and a fall is

[1] Pneumonia, by some modern writers, is classed among the acute specific fevers. It is specific in so far as it is due to a specific virus (the pneumococcus), but since this microbe is capable of causing inflammation of other organs and produces characteristic symptoms only when it affects the lung, and even then not always, since it may produce only broncho-pneumonia, it is more appropriately considered here.

often mentioned as an antecedent of the disease in children. Foul and dusty air and sewer gas are also to be mentioned among causes which may determine the onset of pneumonia.

Morbid Anatomy.

NAKED EYE.	MICROSCOPICAL.
I.–Stage of Engorgement.	
Deep red or purple, very moist on section; crepitates and exudes a frothy fluid on squeezing.	Great dilatation of capillaries of alveoli and bronchioles. Alveoli contain large multi-nucleated cells, derived from the epithelium.
II.—Stage of Red Hepatization.	
Bright red; solid, non-crepitating; friable; granular on cut surface.	Alveoli filled with fibrinous exudation, in which are entangled red and white blood cells and epithelium cells.
III.—Stage of Grey Hepatization.	
Greyish; solid; very friable; yields a yellow pus-like fluid.	Fibrinous exudation broken up by infiltrating cells. When recovery is taking place the cells are large and granular; when destruction, there are more small cells, which infiltrate also the alveolar walls.

All three stages may be present at the same time in the same lung. In children under five years the upper lobe is attacked almost as often as the lower (44 : 100), between five and ten years the proportion is 38 : 100.

The pneumococcus is a small oval microbe occurring generally in couples embedded in an albuminous material, to which the term "capsule" has been applied. It is stained by Gram's method; it grows at temperatures over 24° C., forming on the surface of the culture-medium fine, roundish, transparent masses like dewdrops. It grows rapidly, and when growing in contact with oxygen, reaches its maximum development in about forty-eight hours. Its virulence and vitality thereafter diminish rapidly, and at the end of four or five days it ceases to be possible to start new cultivations. A typical attack of acute pneumonia terminates in a crisis, and it has been found that this coincides with a remarkable diminution in the virulence and number of the pneumococci present in the lungs. This diminution in virulence is attributed to the combined action of the high body-temperature, of phagocytosis, of poisons formed by the pneumococci (antipneumotoxin), and to the production of an acid reaction in the lung, the pneumococcus being unable to develop in acid media.

The *blood* contains an excess of fibrine and an increased number of leucocytes. The leucocytosis corresponds with the temperature, and decreases with it. It is probably related to the phagocytosis which is the main element in bringing about recovery.

The onset of **symptoms** is generally sudden, and marked by shivering, convulsions, or in elder children by a distinct rigor; epistaxis is not uncommon, and there may be severe headache. The symptoms develop rapidly; the most constant is dyspnœa. The respirations rise to 40 or 50, even to 70 or 80, in a minute; and the pulse-respiration ratio is altered. The upper accessory muscles of respiration come into play; the lifting of the upper part of the chest is often very marked, and the associated facial muscles are often thrown into action, causing expansion of the alæ nasi, lifting of the upper lip, and retraction of the corners of the mouth with each inspiration. The pulse at first seldom exceeds 140, but at a later stage it may become uncountable. The symptoms, as well as the physical signs, differ, according as the pneumonia begins at the surface or in a deeper part of the lung; both are more characteristic in the former alternative. Stitch in the side, which is to be attributed to the involvement of the pleura in the inflammation, is present in about half the cases, and renders the cough, which is short and dry, painful, so that the child endeavors to avoid coughing, and often makes a short, grunting sound, apparently due to the suddenness with which inspiration is ended, owing to the pain which it causes. [More commonly the child locates the pain in the abdomen, generally at the epigastrium, and thus may direct our attention exclusively to abdominal trouble if we do not remember this peculiarity.] The skin is hot and dry, and there is a bright red flush over the cheek bones, or over one cheek bone only. This one-sided flush is most often observed in pneumonia of the apex, and generally on the same side as the pneumonia. Sometimes the flush extends over the whole of the face and the upper part of the trunk, and may suggest scarlet fever. Herpes of the lip or chin may precede or accompany the onset of pneumonia, but more commonly it makes its appearance about the second day. The child is often drowsy by day, and restless or delirious at night. It has no appetite, but suffers from thirst, and the tongue is dry and furred, or aphthous. Diarrhœa may come on at the onset of the disease, or at the time of crisis. The liver and spleen are frequently enlarged, and jaundice may be met with, especially in some epidemics.

The temperature rises suddenly, and when the patient is first seen may be 103° to 104° F.; the fever is continuous, and morning remissions are absent or little marked. Defervescence is commonly by crisis at some period between the fifth and eighth days, but the descent of the temperature curve is often more gradual. In either

case the temperature may fall below normal. The crisis may be accompanied by copious sweats, or by diarrhœa. The pulse falls and the dyspnœa diminishes greatly. During the attack the urine is diminished in quantity, and contains less urea and phosphates, and also a smaller quantity of toxins, than in health. At the crisis a very large quantity of urine may be passed, and during defervescence the urine is copious, contains much uric acid, and a large quantity of toxic matters. Albuminuria is not uncommon during the fever, but the amount of albumen is seldom large.

Acute lobar pneumonia, especially when it involves the apex, is, as a rule, attended by congestion of the face, and there is some dulling of the intelligence, but the child can usually be aroused to answer questions. In some cases the onset or the early stage of the fever is marked by convulsions, stupor, or delirium. This is the so-called "*cerebral pneumonia*," but it may be unaccompanied by any gross lesion of the brain or meninges. Convulsions, observed generally in infants, may not come on until the fourth or fifth day, and are preceded by stupor. They vary in severity from irregular movements and some rigidity of the limbs to general epileptiform convulsions. Stupor in its most developed form is seen chiefly in children of two to five years. In older children delirium may be violent, but is usually of the mild and muttering type. Generally both stupor and delirium disappear on the third or fourth day. Ocular or facial paralysis warrants the diagnosis of meningitis, though exceptions occur, and retraction of the neck, though a very unfavorable element in prognosis, is not conclusive evidence of actual meningitis. The physical signs may be very marked or very slight. When well marked there is a slight diminution of expansion with inspiration on the affected side, though the side may be slightly fuller than the other; there is dulness at one base or apex, according to the part of the lung involved; vocal fremitis is increased in this area, and respiration is harsh or bronchial or tubular, with small or fine crepitation at the end of inspiration; the voice sound is well conducted and has a metallic or bleating quality. At an earlier stage there may be no dulness, or the percussion note may be actually tympanitic, while the respiratory sounds are merely feeble, with perhaps a few fine crepitations at the end of inspiration. In such a case the physical signs will probably become well marked within twenty-four hours. In others, again, with symptoms which point strongly to pneumonia, no physical signs of any kind can be discovered in the chest for days. This is commonly attributed to the pneumonia being deep-seated. In doubtful cases search should be directed especially to the angles of the scapulæ, the intervertebral groove, and to the apex. In a well-marked case the breath sounds grow more and more tubular until, when hepatization is complete, the breathing is intensely harsh, or typically tubu-

lar, but unaccompanied by crepitation. When resolution commences crepitations again begin to be heard, and become coarser and moister, until finally they obscure the breath-sounds more or less completely.

Complications.—The serous membranes are specially liable to become infected during pneumonia. The infective agent is generally the pneumococcus, and the inflammation it produces is characterized by the large amount of fibrine in the fluid exuded, so that false membranes are often found; by its richness in cells; and by the bright yellow color of the pus. Occasionally the pneumococcus is replaced by or associated with streptococci or staphylococci.

Pleurisy is an almost invariable accompaniment of lobar pneumonia which reaches the surface. It is characterized especially by the formation of false membranes which vary in thickness, but are commonly thin and soft. As a rule, the quantity of fluid is small, but it sometimes increases as pneumonia subsides. Pericarditis is an occasional complication, commonly in association with pleurisy. The peritoneum may also be involved, the exudation being fibrinous or fibrino-purulent. The joints (especially the knee, shoulder, and thumb) may be attacked, as also the bursa, the inflammation resulting in the production of greenish or yellow tenacious pus. Otitis media is a common complication, though often overlooked. It is frequently double, and causes symptoms which may be mistaken for meningitis. Perforation of the drum will occur in some cases, but symptoms may be relieved by a timely paracentesis. The prognosis as to the recovery of the ear is good, but the risk of mastoid abscess and meningitis is considerable.

Meningitis not secondary to ear disease is a rare complication. *Post mortem* greenish-yellow soft fibrinous exudation is found chiefly along the course of the larger vessels. In many, perhaps the majority of the cases, the meningitis affects the convexity of the brain, and is latent. There may be severe pain in the head, followed by coma, but not infrequently the spinal meninges are also involved, and rigidity and contraction of the neck are observed. Rarely the base of the brain is affected, and paralysis of some of the ocular muscles, facial paresis, and disturbance of the respiratory rhythm may be produced and cause the case very closely to resemble tuberculous meningitis.

The **diagnosis** of acute lobar pneumonia is often difficult at first, owing to the absence of definite physical signs [*e. g.*, in "central" pneumonia]. The onset, in the midst of apparent health, of high fever, with rapid breathing, pungent skin, without any eruption or evidence of acute disease of the pharynx or larynx, and, when these are present, the unilateral flushing of the cheek, and herpes labialis, will raise a presumption of pneumonia; later, the rapid spread of consolidation will increase the probability that the affection is lobar

and not lobular pneumonia, and the occurrence of crisis between the fifth and the eighth day will confirm the diagnosis, and warrant a favorable prognosis. [The atypical course of the disease is fully as common as the typical, and careful daily explorations of the chest are absolutely necessary in obscure cases and will, in time, generally reveal the characteristic signs. This examination should be made with the stethoscope, as with the unaided ear we cannot explore the chest with sufficient minuteness. In addition to the areas already mentioned, we must examine high in the axilla, where we will not infrequently detect diagnostic signs. The signs elsewhere as well as here may be very slight indeed; instead of dulness we may get only a sense of increased resistance to the finger on percussion, more easily detected in children on account of the thin chest-wall; instead of the characteristic bronchial respiration we may hear only respiration slightly higher pitched than that on the unaffected side, at the corresponding point. These two signs have often been made out at the first visit to cases which, twenty-four or forty-eight hours later, showed the dulness and bronchial respiration of a typical lobar pneumonia; taken with other points, the sudden onset, vomiting, elevated temperature, quickened pulse and respiration, they are of great diagnostic importance.

The diseases with which at the outset acute lobar pneumonia may be confused are, acute tonsillitis, scarlet fever, lobular pneumonia, cerebro-spinal meningitis and acute intestinal trouble. Examination of the throat will generally show the characteristic appearance of tonsillitis, and if the case be one of scarlet fever, may show the presence of the enanthem and excite suspicion of this disease. The suspicion of scarlet fever it may be impossible to confirm for a day or two, until the appearance of the characteristic exanthem. The beginning of lobular pneumonia is more gradual and there is, moreover, invariably a more or less extensive bronchitis, generally bilateral. The presence of two or more patches of consolidation points towards the lobular type, yet we must also remember the possibility of "pneumonia migrans," cases of genuine lobar pneumonia not uncommon in children, where the seat of the disease appears to shift rapidly. The exact nature of such cases, it is difficult, at times impossible, to determine clinically. The following case referred to me by Dr. R. H. Babcock, illustrates so well the difficulty of differentiating between these two types of pneumonia that it is given much in detail.

April 27, 189–.

A. B.—Boy, 6 years.

F. H.—Father has gastric trouble.

Previous History.—Cholera infantum at 9 months. "Sensitive stomach" ever since, with frequent epigastric pain after meals. True measles one year ago.

Present Illness.—5 days ago was attacked with rötheln; not at all sick. Now for two days has had considerable cough, without expectoration. No pain anywhere; no apparent constitutional disturbance.

Physical Examination.—Well developed, fairly well nourished, face slightly flushed, respiration rapid but easy; no dilatation of alæ nasi, no expiratory grunt. Slight enlargement of cervical glands on both sides.

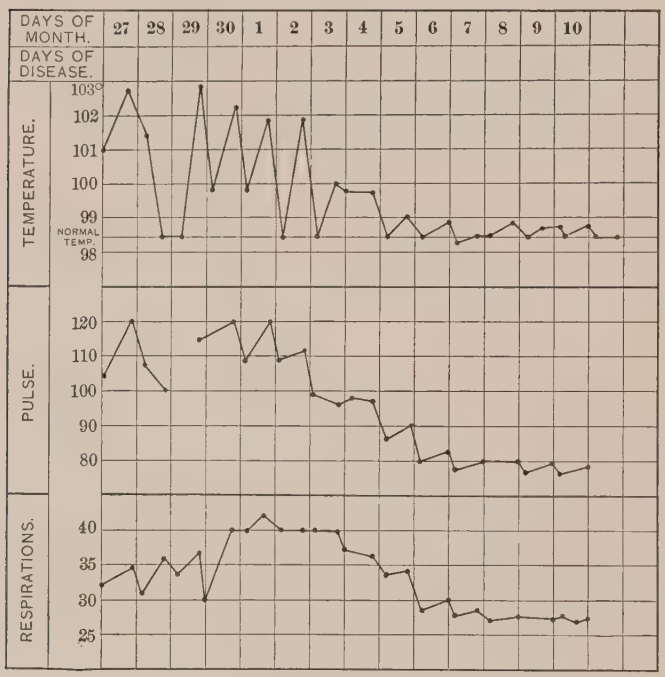
Mouth.—Tongue thin white coat. Throat negative.

Chest.—Lungs negative. Heart negative.

Abdomen.—Negative.

Pulse, Temperature, and Respiration.—Vide chart.

Fig. 52.



Blood { Red 6,104,000, Hb. 65 per cent. } Two hours after breakfast.
{ White, 18,400 }

No plasmodium detected.

Diagnosis (provisional).—Central pneumonia (lobar?) or (lobular?) following so soon after the invasion of rötheln.

April 28th.—Slight dulness and higher-pitched respiration just below left clavicle, as compared with right ditto.

Blood.—Whites, 22,400 (4 hours after breakfast).

Urine.—(24 hr.) 1,000 c.c., pale, acid, 1,015, no albumen, no sugar. Urea, 1.4 per cent., total urea, 14 g. Chlorides 6½ per cent., Phosphates 6½ per cent., Sulphates[1] 1 per cent.

Diagnosis.—Lobar pneumonia, based on temperature and respiration chart, physical signs in chest and diminished chlorides.

April 29th.—Slight dulness and higher-pitched respiration at *left base posteriorly.* Slight dulness at lower angle of right scapula, over limited area.

April 30th.—Signs in left front more marked; scattering medium moist râles on both sides. Backs otherwise same.

May 1st.—Sweating early this A. M. Chest as yesterday. *Sputum* frothy, whitish. Streptococci and tetragenococci. *No* pneumococci.

Urine.—Chlorides 3½ per cent.

May 2d.—Left chest clearing up, top and base. At right base over limited area, between lower angle of scapula and axillary line is dulness, shifting with change in position of patient, and also distant and feebly bronchial respiration (9 A. M.). At 7 P. M. condition same, confirmed by Dr. Babcock, who saw the case in consultation. Aspiration, and about 15 minims of sero-sanguinolent fluid withdrawn.

May 4th.—Right base, respiration more distinct. From this point on the signs in the chest gradually cleared up and patient made an uneventful recovery.

The diagnosis in the above case on the first day was impossible, though the temperature and respiration rate suggested pneumonia, probably lobular on account of the antecedent attack of rötheln. The leucocytosis might have been due to digestion, only two hours after breakfast, or to either variety of pneumonia. The signs on the second day suggested a lobar pneumonia, coming to the surface beneath the left clavicle, those on the next few days a case either of pneumonia "migrans" or lobular with different patches of consolidation, eventually terminating with a pleuritic effusion, a condition more common in lobar than in lobular pneumonia. Bacteriological examination of the sputum showing absence of the pneumococci was against the lobar type. Unfortunately but one examination of the sputum was made, and failure to find the pneumococcus once does not of course prove its absence. The fall of the temperature by lysis, while more characteristic of lobular, is nevertheless observed not uncommonly in lobar pneumonia in children, in whom the course is so often atypical.

The case illustrates the difficulty of differentiating between these two types of pneumonia. We must bear constantly in mind the irregularity of all processes in the young developing organism and

[1] Purdy's method with percentage-tubes and centrifuge. See Chapter XXXVI., p. 414.

hence expect to see atypical cases of diseases, cases not presenting the clear-cut and well-defined course seen in adults.

The symptoms of cerebro-spinal meningitis may at the onset be similar to those of lobar pneumonia. The temperature is lower in meningitis, the pulse is slower and often intermittent, the respiration slower and irregular. Lumbar puncture will be of value in arriving at a diagnosis, the fluid being cloudy and showing bacteriologically the presence of the characteristic diplococci. The subsequent course of the disease and careful exploration of the chest will determine the nature of the case.

Cases of pneumonia beginning with vomiting and diarrhœa, especially in the summer, are sometimes overlooked and regarded as gastro-enteritis; but at the onset the disturbance is too great for intestinal trouble, and, later, examination of the chest will generally show characteristic signs. A marked degree of leucocytosis would point towards a pneumonia.

If convalescence be tedious, the possibility of empyema must be borne in mind. This is unfortunately not at all an uncommon sequel of lobar pneumonia, and should be carefully watched for in all cases with a tardy and slow return to health.]

Pneumonia is not a very fatal disease in children. Holt gives the mortality as 4 per cent.,[1] but it is a little higher under three years of age. High temperature (106° F.) is not in itself of serious significance unless long continued. A sudden rise to 106° F., for instance, is less serious than a continued temperature of 104° or 105° F. In fact, the more acute the onset, and the more rapid the development of recognizable lobar pneumonia, the better, as a rule, the prognosis. Children attacked by well-marked lobar pneumonia are generally of robust type, and it is in such cases that resolution takes place in the most orderly manner. Apart from the occasional occurrence of very high temperature, the chief danger of uncomplicated pneumonia is cardiac failure, either during the height of the fever or at the moment of crisis. Pneumonia of the apex, even if it affect both sides, is of less unfavorable prognosis than in adults—in fact, recovery is the rule. In young children resolution is, as a rule, rapid, but after the age of eight or nine years convalescence is often tedious, and signs of local pleurisy and of imperfect expansion of the lung may remain for many weeks. The chief danger of acute lobar pneumonia in childhood is, indeed, its association with pleurisy.

Pleuro-pneumonia is a more prolonged, more exhausting, and more fatal disease, and owing to the formation of extensive false membranes resolution is more tedious and less complete. The condition is best considered along with other forms of pleurisy.

[1] In 1,482 cases there were 60 deaths. "Diseases of Infancy and Childhood," London, 1897.

The **treatment** of acute pneumonia must be directed to the relief of symptoms. Unless further experience should prove that anti-pneumococcic serum, the use of which is still in the experimental stage, can be relied upon to cut the disease short, no therapeutic means have any effect on the duration of the malady, which is limited naturally. The child should be kept in bed in a room well ventilated and at an even temperature. It should receive small quantities of liquid food at short intervals (every two or three hours). The chest should be enveloped in a closely-fitting woolen garment, or in a cotton-wool jacket. Pain in the chest is most promptly relieved by dry cupping; fomentations, a linseed poultice, or a mustard poultice may be used for the same purpose. Small doses of ipecacuanha combined with sodium carbonate, repeated every hour or two hours for a day or a day and a half, mitigate the feeling of distress and still cough. All sedative remedies are better avoided, but occasionally it may be necessary to give a linctus containing codeine or antipyrin or phenacetin. [In view of the irregular course of pneumonia in children and hence the impossibility of foretelling the hour of crisis, great care should be observed in the use of these antipyretics, lest we get the combined depressing effect of the drug and crisis. A dangerous collapse from such treatment has come under the observation of the reviser. They should always be given combined with some cardiac stimulant: *e. g.*, caffein, coffee or brandy.] Great restlessness is best treated by sponging with cool water, or if the temperature be very high, with cold or iced water. In extreme cases the ice pack to the chest has been used with success, but I have never seen occasion to resort to it. Commencing failure of the heart should be treated by digitalis; at a later stage caffeine or camphor may be of use to tide the patient over the crisis or the hours preceding its occurrence (see p. 79). In extreme cases of distension of the right side of the heart the withdrawal of a few (1 to 3) ounces of blood from the arm is certainly justifiable. After the crisis the patient should be put under the best hygienic conditions available, and should as soon as possible be removed from the room in which the height of the illness was passed. The room should be thoroughly cleansed, and all woolen rugs, etc., as well as articles used by the patient during the illness, disinfected. Change of air should not, as a rule, be advised until convalescence has been completed.

CHAPTER XXVII.

PLEURISY.

Sero-fibrinous Effusion—Purulent Effusion—Symptoms of Pleurisy—Course—Physical Signs—Loculated Empyema—Treatment.

It is customary, in speaking of pleuritic inflammation with a recognizable quantity of fluid in the cavity, to draw a distinction between "pleurisy with effusion," by which is meant pleurisy with serous or sero-fibrinous effusion, and empyema, by which is meant pleurisy with purulent effusion. There is, however, no essential pathological difference, and no greater prognostic difference than there is between the early and the more fully developed stage of any other inflammatory process. This, at any rate, is true in children. If the statement requires any qualification, it is that the prognosis in serous effusion is rather less favorable, inasmuch as it is more often due to tuberculosis. The point is of importance, because it is often assumed, if a first puncture has yielded a serous fluid while a second yields a purulent, that the change was *due* to the puncture, whereas the effusions "are really purulent, and contain the microbic element of pus from the outset, although they appear serous to the eye" (Koplik).

Sero-fibrinous Effusion.—Inflammation of the pleura, with serous effusion only, is less common in children than in adults, and in young children than in those of more advanced age. It is rarely to be recognized in children under two years of age. Rilliet and Barthez, in 341 cases of primary pleurisy in children, found that 101 occurred between two and five years, 111 between six and ten years, and 129 between eleven and fourteen years. The inflammation of the pleura may be primary or secondary.

Exposure to cold is frequently a determining *cause* of pleurisy, and injuries to the chest may also produce inflammation of the pleura with effusion. Pleurisy may be rheumatic, and in some cases appears to be secondary to pericarditis. It may occur also as a complication of typhoid fever, scarlatina, or measles. The most important causes of pleurisy are tuberculosis and pneumonia. Inoculation experiments prove that 40 per cent. of the pleurisies apparently due to exposure to cold are really tuberculous, and clinical observation shows that a very large proportion of patients who suffer from pleurisy with

serous effusion eventually develop tuberculosis of the lungs. The relation of pleurisy to pneumonia is very intimate. In children it is probably the rule to have some involvement of the lung; many writers, indeed, follow the example of Andral, and speak of all non-tuberculous inflammations of the pleura as *pleuro-pneumonia*—a course which is at least extremely convenient, since the diagnosis between commencing pleurisy with effusion and superficial pneumonia accompanied by pleurisy is often difficult and sometimes impossible. Inflammation of the substance of the lung, whatever its extent, is, if it reach the surface, probably always accompanied by some pleurisy. In fact it is not very uncommon for an attack of acute pneumonia in the child to be accompanied by extensive involvement of the pleura, and to be followed by considerable effusion. In broncho-pneumonia the pleurisy is generally local, and the same is true of infarct; but in either case the pleurisy may become general.

Purulent Effusion (*Empyema*).—Pleurisy with purulent effusion is a common disease in children of all ages, but is particularly frequent between the ages of two and six. The effusion may at first be serous, or it may have the characters of pus from the earliest stage. As already said, the difference is one of degree and not of kind. The alteration in the appearance of the fluid is due to an increase in the number of cells—an increase progressive from the first, and dependent upon the character of the inflammatory process.

In the majority of cases of purulent pleurisy in children the pus is thick and viscous—the custard-like pus which used to be called "laudable." Pus of this kind is associated with the presence of the pneumococcus, which appears to be the determining infective agent in 72 per cent. of all purulent pleurisies in children. The purulent pleurisy due to the pneumococcus may be either secondary to pneumonia or pleuro-pneumonia, or primary, although in most cases in which it appears to be primary it is probably either secondary to or accompanied by undiscovered pneumonia or broncho-pneumonia. To the purulent pleurisy which occurs in association with pneumonia Gerhardt, who had recognized the association before bacteriology explained its nature, has applied the term *meta-pneumonic*—a convenient term, since the cases belong to a distinct clinical group which call for a special line of treatment, and in which the prognosis is better than in other forms of pleurisy. The pneumonia may be primary, or may be secondary to scarlatina, measles, or typhoid fever. The pleuritic complication may be discoverable during the pneumonia, soon after the crisis, or some weeks or even a month later. The symptoms of onset may be well marked or latent, and the pleuritic process may terminate in absorption, by opening into a bronchus, by becoming encysted, or by opening externally, perhaps after tracking far down, so as to point in the lumbar region or groin.

In a minority of cases in children purulent pleurisy is associated with the presence of one of the ordinary pyogenic cocci, and in a considerable proportion of the cases the primary lesion is tuberculous. The disease can be shown to be secondary to some contiguous local centre of inflammation in the majority of cases—broncho-pneumonia, suppurative angina, perhaps pericarditis or peritonitis. In a few it occurs in the course of scarlatina, diphtheria, erysipelas, or otitis, and in these cases the infective agent is probably carried by the blood. In a few cases only can no primary purulent focus be discovered. Puncture at an early stage may withdraw a clear fluid, which, however, gives a slight precipitate on standing. At a later stage, and in some cases at the earliest stage at which exploratory puncture appears justifiable, the fluid is purulent. It is a thinnish, yellow pus, which quickly lets fall a fine powdery precipitate.

The **symptoms** of pleurisy vary very much in intensity. Small patches of localized pleurisy associated with broncho-pneumonia are difficult to recognize, since the physical signs do not present any marked peculiarities, and complaint of pain in children is often very indefinite. The degree of pain appears, indeed, to be very different in different cases. In children of six or seven to twelve years old, attacks of acute dry pleurisy, attended by much pain, are not uncommon. Friction may be absent, unless the child takes a deep breath. Deep inspiration brings out a dry, creaking, or fine crepitant râle, which may be heard only at the end of inspiration. Expansion on the affected side is diminished owing apparently to the pain which a full inspiration causes. In a well-marked case the child sits with the shoulder on the affected side lower, nursing the elbow against the chest. Owing to the deficient expansion the breath-sounds on the affected side are feeble, and it may be difficult to induce the child to take an inspiration deep enough to elicit friction. The percussion note is not altered. The general symptoms are not very distinct; the temperature may be very little above the normal, but it may touch 100° or 101°, the pulse is quickened a little, and there is some malaise and perhaps headache and loss of appetite. In such a case the symptoms described may all subside in a few days or they may be succeeded by signs of effusion. The effusion leads to a diminution or disappearance of pain, and a false appearance of general improvement. It is, therefore, unwise to trust to the patient's sensations, and very desirable to make a careful physical examination before accepting the patient's assurance that he is cured. Some of these cases are probably rheumatic.

The onset of the severer form of pleurisy may be acute or insidious. If *acute*, it resembles the onset of pneumonia, which, indeed, is generally co-existent. The disease begins with a rigor, or shivering and a sensation of chilliness in older children, with stupor or convulsions

in younger. The temperature rises to 103° or even 105°, the pulse to 140 or even 180, though when these extremes are reached there is probably a good deal of pneumonia also; respiration is hurried and shallow, and on the affected side expansion is diminished. The expression of the face is anxious or peevish, cough if present is short or painful, so that it is often followed by crying. Children of five or six years, or even younger, may point to an area where pain is felt. This area will be a little tender, and on deep inspiration friction or a small crepitant râle may be heard. If the onset be *insidious* the child is brought under treatment because it is pale and languid, has a slight dry cough, and complains of pain in the side or abdomen (generally the epigastrium). The pulse is a little hurried, as is respiration, and the child is found to have some irregular fever. When the disease has become *established*, and there is more or less effusion, there is marked pallor of countenance, perhaps some cyanosis, though this is less than in pneumonia, dyspnœa, and some pain or discomfort in the chest. The general condition of the patient deteriorates quickly, and there is rapid emaciation. The temperature is high, with remissions which generally occur in the morning but seldom reach normal. Pain in the side is generally less than at an earlier stage, but tenderness may be more pronounced. When well marked it probably indicates that the fluid is becoming more distinctly purulent.

In **pleuro-pneumonia**—when, that is to say, there is extensive infection of the lung and pleura by the pneumococcus—the signs of consolidation will be marked early, and an imperfect crisis will occur on the eighth, ninth, or tenth day, or even later. Defervescence is not complete, and fever remains, of hectic type but with remissions at irregular intervals.

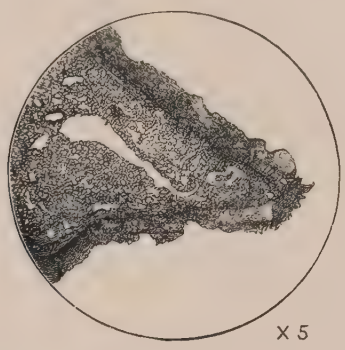
The *termination* of an attack of pleurisy may be in resolution without effusion, but with the formation, probably, of adhesions, which may be the source of after pain. If fluid is effused, it may become purulent; this is in children by far the most common termination. In many cases the surface of the lung is from an early stage covered with false membrane (Fig. 53), and eventually a condition may be produced to which the term *thickened pleura* is commonly applied—a condition in which the false membranes persist. They become œdematous, and there is also perhaps some free fluid, generally purulent. The membranes in time become more or less organized, and there is accompanying fibroid degeneration of the pleura and subjacent lung.

The recognition of the purulent nature of a collection of fluid in the chest is often exceedingly difficult until an exploratory puncture with a hypodermic syringe has been made. The more acute the history of the case, the more likely is it that the fluid is purulent. That

the attack has come on during scarlatina, measles, or some other exanthem is evidence in favor of fluid. Tenderness, œdema, or localized redness also point to pus, the last-named sign perhaps to approaching pointing of the pus at the surface. The duration of the case must also be taken into consideration; if the history extends to three or four weeks, and there are signs which point distinctly to fluid rather than to thickened pleura, the diagnosis of pus may be made with considerable confidence, especially if the temperature is of the hectic type, and the patient losing weight rapidly.

The *physical* signs of fluid in the pleura are the same whatever its nature, but they vary according to its amount and situation. When the quantity of fluid is large there ought to be no difficulty in making a diagnosis, although mistakes are often made. It should never be forgotten that a case which begins as pneumonia may terminate

FIG. 53.



Section of the border of a lung affected by acute pleuro-pneumonia, showing the thick false membrane. × 5. (From photomicrograph by Mr. F. FOWKE.)

as one of effusion into the pleura. On inspecting a chest in which a considerable quantity of fluid is effused into one or other pleural cavity, it is seen that there is less movement on the affected side. When the quantity is large there may be some bulging of the chest as a whole, though the intercostal spaces may not be full, may indeed even recede in inspiration. There is loss of vocal resonance, which, however, cannot be perceived unless the child cry, which it is often indisposed to do, owing probably to the pain thereby caused. Alteration in vocal fremitus cannot, for the same reason, be depended on to give much information. Palpation may detect some fulness of the intercostal spaces, some defective expansion not observed by the eye, but the most important information it can afford is as to displacement of the heart. If the effusion is on the right side, the heart may be much displaced to the left, so that the apex beats outside the nipple,

or at the axillary margin. When the effusion is on the left side the displacement of the heart is not so easily perceived unless the quantity of fluid be large. In a case in which old pleurisy and fibroid phthisis can be excluded, well-marked displacement of the heart is almost conclusive evidence of fluid in the pleura, but failure to detect displacement does not negative a diagnosis of effusion rendered probable by other signs, since the quantity of fluid may be too small, or adhesions may retain the heart near its normal position. Moreover, the heart-beat may be difficult to localize or to perceive at all by the hand, and then reliance must be placed on an estimate of the point at which the heart-sounds are best heard. The percussion note over fluid is quite dull, and a peculiar sensation is communicated to the finger in contact with the chest, which is commonly described as "wooden." This peculiar woodenness is characteristic, and the best single sign of fluid; but if the percussion be too heavy it may not be perceived, and a resonant note may be obtained in the elastic chests of young children, owing apparently to the resonance of the lung of the opposite side. A light, short percussion stroke is therefore necessary. Immediately above the upper level of the fluid a tympanitic note may be obtained, probably produced by relaxed lung, but this will not be observed in the later stages of the case, when the lung is completely collapsed in this area. It will seldom be possible to make out that the dulness shifts with change of attitude; indeed, the constancy of the area of dulness in all attitudes and often for many days together is remarkable. The signs of dulness will probably first be detected at the back, and the dulness will reach higher in the vertebral groove near the spine; later, the vertical depth of dulness is often greatest in the axilla. Very little information can be obtained from auscultation, especially in the later stages of fluid effusion into the pleura; the breath-sounds over the dull area may be bronchial, or even tubular, in which case they appear to confirm a diagnosis of pneumonia, or they may be feeble or absent, as in the earliest stage. When the quantity of fluid is large the breath-sounds, whatever their tone, are, as a rule, feeble and distant. The percussion note over thickened pleura is dull, high-pitched, but not wooden.

Loculated Empyema.—An important class of cases remain for consideration—those in which the fluid is from an early period limited by adhesions. The fluid which is nearly always purulent, may be localized at any part of the surface of the lung—back, front, axilla, or apex; or it may gather between the lobes—the so-called cystic empyema; or it may accumulate between the base of the lung and the diaphragm—diaphragmatic pleurisy. On the surface the symptoms and signs are those of ordinary pleurisy, except that they involve a limited area, and are therefore particularly liable to be

mistaken for pneumonia or broncho-pneumonia. A certain diagnosis is not to be made without exploratory puncture, unless the quantity of fluid be so large as, practically, to take the case out of the category of loculated empyema, which we are now considering. Interlobar pleurisy (cystic empyema) can seldom be diagnosed with certainty. The diagnosis of diaphragmatic pleurisy is exceedingly difficult and uncertain. The symptoms are very obscure. Pain is referred to the upper part of the abdomen; on the right side there may be notable depression of liver, but as there is dulness at the base back and front due to the fluid, it is not easy to say that enlargement of the liver may not be the cause of its lower border being unduly low in the abdomen. As a matter of fact, diaphragmatic pleurisy is seen more often in the *post-mortem* room than diagnosed during life, except in those rare cases in which the pus tracks down and points in the loin or groin.

The **treatment** of acute pleurisy must be symptomatic. A dose of calomel given at the onset is beneficial in most cases. In the early stage, when there is violent stitch in the side, sodium salicylate gives relief sufficiently often to render it worthy of trial, the more so that in some cases it appears to check the effusion of fluid also, and so to bring the attack to an end. When this drug fails the pain may be relieved by hot fomentations, or by the local cold pack, or, if necessary, by the ice poultice. Iodine and other counter-irritants seldom succeed, and are not desirable applications in young children. When the amount of fluid effused becomes considerable, the pain is greatly diminished; if the temperature subside simultaneously, and the breathing be not greatly embarrassed, no active treatment is required. The patient should be kept at rest but not confined to the house, the bowels should be caused to act freely every day, and light food should be given. If the quantity of fluid effused either at the onset or later be sufficiently large to embarrass respiration and to produce, perhaps, some cyanosis or anasarca, there should be no hesitation in drawing off the fluid with antiseptic precautions. There remain a class of intermediate cases in which it is often very difficult to decide whether aspiration is advisable. The operation itself, if the fluid be withdrawn gradually, and if antiseptic precautions be observed, is harmless, and it is certainly inadvisable to postpone it too long; the withdrawal of part of the fluid is sometimes followed by absorption of the remainder. If exploratory puncture shows that the fluid is purulent, incision followed by drainage is usually necessary, and should not be long deferred.

[Immediate evacuation of the fluid is imperative as soon as a diagnosis of empyema is made. Delay is dangerous, and the earlier the operation, the better the results. The pus should be evacuated by either aspiration, simple incision, or resection of a rib. As to

which of these methods should be pursued must depend on the circumstances of the individual case. The general condition of the patient and the type of pleurisy present must be taken into consideration. If the child be in a weak cachectic condition, as so often happens in hospital cases coming in late in the disease, simple incision is to be preferred. Later, after the child's general condition has improved by tonic treatment, it may be necessary to resect a rib. In uncomplicated pneumococcic empyema, simple incision or even aspiration is generally sufficient, but in cases of streptococcic empyema, a free opening with resection of a rib is best. A bacteriological examination of the fluid obtained by exploratory puncture is thus, it will be seen, of practical importance in deciding upon the choice of operation.]

CHAPTER XXVIII.

CHRONIC AFFECTIONS OF THE BRONCHI.

Chronic Bronchitis and Emphysema—Bronchiectasis—Asthma—"Hay Fever."

Chronic Bronchitis.—Children who have had one attack of acute bronchitis are not only predisposed to fresh attacks but in many cases suffer in the intervals from chronic bronchial catarrh. After measles and whooping-cough this tendency is particularly marked. The subjects are generally anæmic and ill nourished, the skin puffy, the muscular system flabby. If under three years old, they are usually rickety, and often suffer from gastro-intestinal catarrh. Cough is troublesome, especially at night, often paroxysmal. The chest is resonant on percussion, and the note often high-pitched over the upper parts of the lungs in front. A few scattered sibili may be heard, or coarse rhonchi, in the interscapular region. A tenacious clear mucus is expectorated, if the child be old enough, in small quantities. On exposure to cold, or any slight disturbance of the general health, such as may be produced by dentition, an aggravation of all the symptoms is very apt to occur, and numerous sibili and rhonchi of various degrees of coarseness may then be heard. The expectoration becomes more abundant and fluid. Later it is muco-purulent and the cough loose. The mucous membrane of the bronchi is hyperæmic and thickened, and there is usually some emphysema of the apices and anterior borders of the lungs. As a rule, except during the exacerbations, respiration is free, but in children over six or seven years old attacks of dyspnœa of the nature of spasmodic asthma may occur.

Typical examples of *chronic emphysema* are not very uncommon in children above the age of nine or ten years. The patients, usually boys, have a characteristic appearance. They are clumsy and thickset in figure, slow in movement, and indisposed to exertion; the shoulders are rounded, and they stand with the head bowed and the arms hanging loosely; the face is broad and congested, the chest barrel-shaped, the bones of the limbs are large, the fingers clubbed, the skin dry and harsh; the chest is sub-tympanitic on percussion, the breath-sounds are weak generally but rustling at the apices; the appetite is poor, the bowels costive. Slight exertion causes dyspnœa, which is often accompanied by some rhonchus, and followed by

paroxysmal cough, ending in the expectoration of frothy mucus. Attacks of an asthmatic character may occur without obvious cause. The patients are extremely liable to suffer from bronchitis. The dyspnœa is then much increased, they are unable to lie down, and the congestion of the face becomes extreme. Fits of coughing are severe, and end often in vomiting. At the beginning of one of these attacks, loud sonorous and sibilant rhonchi generally obscure all other sounds, and expectoration is scanty and tenacious. After a day or two small rhonchi and loose mucous râles are heard in all parts of the chest. The temperature is a little raised. During the winter many such attacks occur, and the patient is never really free from bronchitis except during mild, warm weather. With each winter his condition grows worse. He becomes much emaciated, the right side of the heart dilates, and the dyspnœa on slight exertion confines him to a sedentary life. Chronic gastro-intestinal catarrh is a common complication.

The history of the case and the physical signs usually render the **diagnosis** of chronic bronchitis easy, but it should be remembered that a large proportion of the cases to which this term is popularly applied are really examples of granular pharyngitis or adenoid disease of the naso-pharynx (*q. v.*). The diagnosis of spasmodic asthma should only be made after bronchial catarrh, and enlargement of the tracheo-bronchial glands have been excluded. This will not often be the case, though in many cases of chronic bronchitis a spasmodic element in the production of the attacks of dyspnœa may be recognized.

The **prognosis** in simple chronic bronchitis depends far more upon the general state of the patient's health than on the local condition. Chronic bronchitis in a rickety child is always a rather serious condition, owing to the fact that it favors collapse of lung and the occurrence of acute bronchitis and broncho-pneumonia. In older children the degree of emphysema is the most important element in prognosis. When this is well marked, when there is dilatation of the right side of the heart, and cyanosis even though slight, there is little prospect of recovery, and few such patients reach adult age. Each winter sees an aggravation of their condition, and they succumb to intercurrent acute bronchitis, with which, owing to the embarrassment of the heart already existing, they are little able to cope.

In the **treatment** of chronic bronchitis in young children the most important points are to prevent fresh attacks of acute or subacute bronchitis, and to improve the general nutrition. Cod-liver oil is very valuable in these cases, especially when, as commonly happens, the child is rickety. The ordinary expectorants are not of much use, but Dr. Eustace Smith recommends liquid tar (one drop two or three times a day on sugar, or in older children in capsule or pill).

Counter-irritants to the chest are useful. Turpentine liniment well rubbed in is the best, since some turpentine is absorbed and has a beneficial action. Iodine liniment is also a good application, different areas, each about three inches in diameter, being painted daily. The child should be clothed in woolen garments next the skin, and if, owing to wet and damp weather, it be necessary to keep it indoors, gentle gymnastic exercises are advantageous. When possible, it should pass the winter in a warm and sunny climate, so that it may be able to get out of doors daily. In marked cases of emphysema in older children treatment by drugs has little effect. When cough is troublesome stimulant expectorants and turpentine inhalations by day, and turpentine liniment rubbed into the chest at night, are useful. Gentle gymnastic exercises on the Schott system are of use if there be cardiac dilatation. Winter in a cold damp climate entails much confinement to the house, and attacks of bronchitis are almost impossible to be avoided. A mild, sunny, and rather moist climate suits these patients best.

Bronchiectasis.—The frequency with which dilatation of the bronchi occurs in children is a point upon which there is room for much difference of opinion. In a large proportion of cases it is associated sooner or later with tuberculosis. It cannot be produced by mechanical means alone, as, for instance, by coughing. Some lesion weakening the bronchial wall must precede the dilatation.

In children the most important causes are broncho-pneumonia, especially that secondary to measles, whooping-cough, influenza, and typhoid fever; pleurisy and pleuro-pneumonia; and, more rarely, primary chronic tuberculosis of the lungs. Among the less common causes may be mentioned chronic bronchitis, atelectasis, foreign bodies in the bronchi, constriction of a bronchus, and pulmonary syphilis. Any debilitating general condition, such as malnutrition, overcrowding, rickets, or chronic enteritis, tends to render permanent the dilatation which accompanies broncho-pneumonia. The mode in which these many causes bring about dilatation is different. In broncho-pneumonia the small bronchi in the affected areas share in the inflammation. Their walls are weakened, and, the surrounding alveolar tissue being either consolidated or collapsed, the positive pressure in expiration or during cough must act upon the bronchi and upon the adjacent unaffected lung tissue, producing in the one case bronchiectasis and in the other alveolar emphysema. Partial obstruction of a bronchus impeding complete expiration will favor the dilatation of its distal part. The dilatation thus produced is doubtless, as a rule, transient, and a gradual restitution accompanies the recovery of the lung. Godlee[1] has called attention recently to the advisability in cases of unilateral bronchiectasis, in children es-

[1] Roy. Med. Chi. Soc., March 24, 1896.

pecially, of making careful inquiries as to the possibility of a *foreign body* having been inhaled. In some cases such an accident may cause acute bronchiectasis and gangrene of the lung, but in others the inconvenience produced at the time is so slight that the accident is forgotten, or not connected with the subsequent pulmonary disease. If the foreign body be expelled, (1) complete recovery may take place, (2) the signs of bronchiectasis may persist, or (3) the patient may succumb to tubercle engrafted on the primary bronchial disease.

To produce permanent bronchiectasis it is necessary that fibroid changes should take place in the lung. Thus if the broncho-pneumonia be tuberculous, or become so, the sclerosis which attends the retrogression of the tuberculous process may render the bronchial dilatation permanent and in time increase it. Pleuro-pneumonia and pleurisy leading to extensive adhesions or to long-standing collapse of lung, with subsequent imperfect expansion of the alveolar structure, as in empyema, are always attended by dilatation of bronchi. Under such circumstances, when the chest wall and the thoracic and abdominal organs have been displaced as much as possible, the contracting forces tend to produce dilatation of the bronchi. The bronchi most often dilated are those of medium and small size, and the changes are limited usually to one lung, and during childhood to the base. When established, the bronchiectasis is usually fusiform, but it may be cylindrical or form a lateral dilatation. The bronchus on the proximal side of the dilatation may be of normal calibre or contracted; beyond it may be obliterated. The pulmonary tissue surrounding the dilatation is sclerosed, and the scattered areas of emphysema in the parts of the lung which still remain pervious. Occasionally a single bronchus presents several dilatations. The lining of the dilatations is formed by a degenerate mucous membrane, which in chronic cases may have a granular surface. In association with decomposition of the retained secretions, superficial gangrene of the mucous membrane may occur. The communication between the proximal end of the bronchus and the dilatation may become closed, and a caseous and calcareous degeneration then ensues. The dilatation of bronchi secondary to empyema is commonly extensive, several or many bronchi being affected in their whole extent.

The **physical signs** vary according to the extent and degree of the dilatation. When broncho-pneumonia is complicated by considerable bronchiectasis there will be found at the posterior bases, but more marked usually on one side, and generally within an area of deficient resonance, increased vocal resonance, cavernous or even amphoric respiration, and coarse metallic or loose bubbling râles. In chronic bronchiectasis with pulmonary sclerosis the physical signs vary according to the nature of the condition upon which the dilatation de-

pends. If on chronic bronchitis, which leads to dilatation of the large bronchi, there will be loud bronchial and even cavernous breathing in the interscapular region and below; if on old pleurisy, there will be the deformities produced by that condition, but the sounds produced within the dilated tubes will depend on whether they be full or empty. "If the dilated tubes are full of muco-pus, the breath-sound is weak and bronchial, with little rhonchus; and the resonance of the voice when the child speaks is faint or suppressed. If the air-passages are comparatively empty, the respiration is loud and blowing, often intensely cavernous, or even amphoric, with metallic echo; and large, crisp, metallic bubbles, with dry creaking sounds, are heard with both inspiration and expiration." [1]

The **symptoms** produced by bronchiectasis are governed to a great extent by the condition to which it is secondary. When extensive, the characteristic symptom is the sudden onset, generally in the morning, of a severe paroxysm of coughing, producing much distress and congestion of the face and ending in the expectoration, often accompanied by vomiting, of a large quantity of sputum, of a grey or grey-brown color, as a rule fluid, and often, owing to retention, very offensive. On standing, it separates into a granular puriform layer below and a mucous layer above, upon which float muco-purulent shreds and a brownish froth. The respiration is a little hurried. In time considerable dilatation and hypertrophy of the heart may be produced, with some constant cyanosis of the face and clubbing of the finger-tips. Bronchiectasis may cause little or no fever; but in time, if the dilatations are numerous and large, the patient begins to suffer from hectic fever, and becomes much emaciated. In the majority of cases this unfavorable change is due to intercurrent tuberculosis; but it is aggravated, and in some cases produced, by the absorption of toxic bodies from the retained secretions. On the other hand, bronchiectasis may persist for many years, from childhood to age, without preventing the sufferer from following an active life, and recovery, though rare, is not impossible, if the dilatation be not too considerable. On the whole, therefore, the *prognosis* depends rather upon the nature of the complications than upon the mere discovery of bronchial dilatation.

The **diagnosis** is often a matter of great difficulty, and is commonly impossible unless the patient can be watched for some time. The special characters of bronchial dilatations are that they are usually situated at the bases, and that they are stationary—that is to say, the physical signs they produce show no tendency to spread over a larger area, but rather the reverse. Confirmatory evidence that the process is not tuberculous is afforded by the absence of fever, and of tubercle bacilli from the sputum.

[1] Eustace Smith, "Disease in Children," p. 502.

Treatment directed to the relief of the symptoms produced by bronchiectasis is seldom called for, except when the expectoration is foul and copious, the two conditions going usually together. Of all internal remedies turpentine is the best. It diminishes the amount of secretion and checks the tendency towards decomposition. The oil of turpentine may be given to the extent of ℳx–xx daily to a child of 10 or 12 in three doses, and the air of the room may be charged with it. Various substitutes have been suggested, such as pure terebene, and terpine, and the oils of fir and of eucalyptus for diffusion through the air of the room. Inhalations of creasote, carbolic acid, etc., are not of much use, but as an internal remedy creasote is often useful. Tar administered in capsules, pill, or the syrup of tar (U. S. Ph.) often gives relief. The cough should not be checked unless it interferes with sleep. Frequently the patient is aware that a particular attitude—generally lying down on the side on which he does not habitually sleep—will excite the cough; and when this is the case, he should be advised to assume this position twice or thrice in the twenty-four hours so that the dilated bronchi may be emptied. Intratracheal injections (menthol 10 per cent. and guaiacol 2 per cent. in olive oil) have been recommended for adults, and incision of the cavity followed by drainage has given good results in some cases. The general health should be maintained, and a mild winter climate is desirable, so that the patient may be able to spend much time out of doors. A dry is better than a moist climate, except in very extreme cases.

Asthma.—"Bronchial asthma is a neurotic affection, characterized by hyperæmia and turgescence of the mucosa of the smaller bronchial tubes and a peculiar exudate of mucin" (Osler). Accepting this definition, true asthma must be held to be uncommon in childhood, but children over four and five years of age, who are the subjects of chronic bronchitis and emphysema, or adenoid vegetations and obstruction of the nose, are very liable to asthmatic attacks, which may be brought on by sudden exposure to cold, by air laden with dust, or by dyspeptic disturbances.

During the *attacks* the breathing becomes labored, expiration prolonged, and the chest full of sibili. The face is flushed, and expresses much distress. After a few hours the difficulty in respiration passes off, but the child is exhausted, and dreads a recurrence. At the end of the attack expectoration, previously suppressed, becomes free; it is accompanied by cough, and often contains small masses of thick tenacious mucus. In its more marked forms bronchial asthma is probably in most cases due to a chronic relapsing bronchial catarrh characterized by the formation of a plastic exudation. It is this exudation which yields the tenacious masses above mentioned. In rare instances the masses may have a distinct dendriform shape, apparently casts of the smaller bronchi.

Children are liable also to **hay fever**. The attack begins with itching of the nose and coryza. Usually some bronchial catarrh ensues, and the child loses appetite and becomes restless and irritable. Only in rare cases are distinct asthmatic attacks observed.

In the **treatment** of children liable to asthmatic attacks attention should first be directed to the relief of bronchial catarrh or naso-pharyngeal disease, if these be present, and to the prevention of dyspepsia by careful dieting. When bronchial catarrh is present, the addition of potassium iodide to an expectorant mixture may ward off the attacks. During the attack nitre paper may be burned in the room, or in very severe cases four or five drops of chloroform may be inhaled from a handkerchief. The best climate for patients subject to asthma, if not also suffering from emphysema, is that of a high and dry health resort, such as the Alps in summer, though hay and dust should be avoided. In the treatment of hay fever the use of antiseptic lotions (boric acid, perchloride of mercury 1 in 4,000) for the eyes and nose at the onset may arrest the attack. A bland ointment (such as borax lanoline) should be introduced into the nose. As a rule, however, such patients do not do well in the country in the spring and early summer, and should, if possible, live in a town or at the seaside.

CHAPTER XXIX.

PERITONITIS.

Acute Peritonitis—Chronic Peritonitis—Appendicular Peritonitis; Local Adhesive Peritonitis; Perityphlitic Abscess; Acute General Peritonitis.

If cases due to tuberculosis be excepted, peritonitis, whether acute or chronic, is a rare affection in infants and children.

Acute peritonitis occurs sometimes in new-born infants as a consequence of septic infection. In older children it is a consequence in the majority of cases of inflammatory disease of the vermiform appendix. Among the remaining cases to be enumerated the most important is injury; others are intussusception and foreign bodies or fæcal masses impacted in the intestine. It has been observed also as a complication of certain infectious diseases—small-pox, diphtheria, typhoid fever, and especially scarlet fever. The inflammation is associated with the presence of certain microbes, among which may be mentioned as the most common the bacillus coli communis, but streptococci and the diplococcus pneumoniæ have also been found.

The **morbid appearances** in the peritoneum are vascular injection, ecchymoses, and fibrinous exudation. The amount of fluid effused is usually scanty, but may be very considerable. It seems to be more liable to become purulent in children than in adults.

The **symptoms** are the same as those in the adult. The patient lies on the back with the knees drawn up, and dreads every movement which aggravates the severe abdominal pain from which he suffers. The belly quickly becomes distended, tense, and tympanitic, the respiration thoracic. Dulness on percussion may be discoverable in the flanks or iliac fossa, but this is the exception. Thirst is intense, but everything which is swallowed is rejected almost immediately. The tongue is small, dry, and red. Constipation is almost invariable, and retention of urine not uncommon, owing in both cases to paralysis caused by the peritonitis. Fever is high, the skin hot and dry, the pulse rapid and hard, and after a short time thready. Death occurs in a large majority of all cases within four to eight days, but if the patient survive the intense nervous depression and general exhaustion accompanying the onset of the disease, immediate recovery is possible. More often in children suppuration occurs; the acute symptoms subside to some degree, and then, as a rule dur-

ing the second week, the fever returns, accompanied often by a rigor; the belly increases in size; dulness and perhaps fluctuation becomes perceptible; the navel becomes everted, red and tender, and finally gives way, affording exit to a large quantity of purulent fluid. In other cases the suppuration is more limited, and makes its way towards the surface at any point, but most often in the hypogastric region; the skin becomes red, and fluctuation may be perceived. Spontaneous rupture through the umbilicus, or evacuation of a localized collection of pus by incision, appears generally to be followed by recovery.

The **diagnosis** as to the cause of the peritonitis, upon which the **prognosis** must in a large measure depend, is generally very doubtful, except when the peritonitis is due to inflammation of the appendix. It is seldom possible to exclude tuberculosis, which is by far the most common cause of acute peritonitis in children.

The **treatment** of acute peritonitis can be palliative only, unless it be considered prudent to follow the method recommended by some abdominal surgeons of giving saline purgatives when the onset of peritonitis is apprehended after laparotomy. Under ordinary circumstances, cases will hardly be seen early enough to render such a course of treatment otherwise than certainly disastrous, and it is contraindicated in perityphlitis, which is the commonest cause of acute peritonitis in childhood. Hot or iced applications to the belly, if applied carefully and lightly, give relief to the pain. The propriety of prescribing opium or morphia has given rise to much difference of opinion. It is sometimes impossible to refuse the patient the relief which it promises, but weight must be given to the argument advanced by surgeons that by its use the symptoms are masked. If the propriety of laparotomy is under consideration, opium should not be given until the patient has been seen and examined by the operator, and a decision has been come to either for or against surgical interference. In coming to this decision there ought to be as little delay as possible. There is, however, no objection to giving relief even to the extent of masking the symptoms, if an accurate diagnosis of the cause of the peritonitis has been made.

Chronic peritonitis not due to tuberculosis is a very rare affection. Since the possibility of recovery from tuberculous affections has been more generally recognized its existence has been denied, and it is certainly exceedingly difficult in the great majority of cases to exclude tuberculosis. It is an occasional consequence of acute traumatic peritonitis, and a rare sequel of measles. It may be produced by diseases of the spleen and liver, especially hydatid, by typhlitis, by chronic heart disease. Hepatic cirrhosis must also be enumerated among the causes of chronic peritonitis. When due to disease of one of the abdominal organs, it is, at first at least, localized. Cases are met with

occasionally, especially in girls at puberty, but also in young children, in which considerable ascitic effusion occurs without obvious cause, and it is usual to attribute the effusion to chronic peritonitis, which is then termed idiopathic. The term is, however, a confession of ignorance. After the fluid has been absorbed, or perhaps drawn off on more than one occasion, the belly becomes tense, and generally resonant, but patches of dulness remain, and hard masses may be felt; these are supposed to be due to thickening between adjacent adherent coils. The majority of such cases are without much doubt tuberculous. When due to injury or to disease of one of the abdominal organs, chronic peritonitis is at first localized and plastic. The amount of fluid effusion is small, but the tendency to adhesion is marked. In this way organs may be bound down in abnormal positions, and bands formed which may eventually be the cause of internal strangulation. The great omentum may become deformed and retracted.

The **symptoms** of chronic peritonitis vary with the nature and extent of the lesion. In chronic peritonitis after measles, and in the "idiopathic" form, the first thing to attract attention is the enlargement of the abdomen, which is found to be due to fluid. In these, and in the traumatic cases also, it may be impossible to elicit any history of pain or evidence of tenderness. In local peritonitis local pain and tenderness with increased resistance, or an ill-defined tumor, may be present, but it appears certain that in many cases the symptoms are so slight that medical advice is not sought.

The effects of **treatment** directed to the local condition are not, as a rule, well marked. Counter-irritation with iodine may be tried, one-quarter of the abdominal surface being painted with tincture or liniment. Painting with iodoform collodion has been recommended, and advantage sometimes appears to be derived from the use, for a week or ten days, of mercurial ointment rubbed gently into the belly and then covered with a bandage. In any case, but especially when there is much ascites, a broad flannel bandage should be firmly applied next to the skin, and worn night and day, and the thighs and legs should be kept warm. When the amount of fluid effused is large enough to distend the abdomen the operation of paracentesis should not be too long delayed, and should be repeated, if necessary, without hesitation. Should the fluid withdrawn be purulent, it can hardly be doubted that an exploratory laparotomy ought to be performed with the view of evacuating the pus, which will probably be contained not in the general peritoneal cavity but in a part limited by adhesions. Chronic peritonitis, however, attended by suppuration, is, in very many cases, tuberculous. Internal remedies, such as potassium iodide, have little or no influence on the local condition, and exercise a depressing effect on the general health. The main point to be kept in

view is to maintain the general nutrition by supplying easily digested food, especially milk, meat (including chicken and fish), and fats (butter and bacon).

Appendicular Peritonitis.—The vermiform appendix is in man an obsolete and functionless organ; its length, in proportion to that of the large intestine, is greater in the infant (one to ten) than in the adult (one to twenty). Its actual length is about 1 in. at birth, about 3 in. at five years old, and about $3\frac{1}{2}$ in. at ten years old; it attains its maximum length (under 4 in.) before the age of twenty. Its position varies. In about one-third it runs up along the left border of the cæcum, in another third it lies either behind the cæcum or in intimate relation with it. In about one-sixth it hangs down into the pelvis, and in a few it runs transversely across the psoas and may reach the left side.

It is liable to inflammation of its mucous and other coats, and this liability is greater under twenty years of age than above. In the series of cases recorded by Hawkins[1] at all ages from five upwards, 11.6 per cent. occurred between five and ten years, and 43.3 per cent. between ten and twenty, but it may occur in infancy, and is altogether probably more frequent in childhood than is generally recognized.

Catarrhal inflammation is probably common, but produces no symptoms unless the peritoneal coat become involved. It may end in (1) recovery. (2) In obliteration owing to shedding of the epithelium; a granulating surface is produced which leads to complete occlusion throughout its whole extent, or a limited stricture followed by cystic dilatation of the part beyond. This is rare in children. (3) A chronic condition, with great thickening, so that the appendix cannot collapse and become obliterated, but remains patent, secreting pus.

Ulceration may be a consequence of catarrh, or may be produced by a fæcal concretion, far more rarely by a foreign body, such as the traditional cherry stone. The appendix, especially in children, appears usually to contain some fæces, but under pathological conditions a small mass may be retained, and in time become coated with lime salts. The ulceration may lead to peritonitis and perforation.

Infective appendicitis may be primary or may succeed catarrh or ulceration. It is an acute inflammation involving all the coats, and may begin apparently either in the mucous membrane or in the submucosa. It may affect a larger or smaller area, and may end (1) in necrosis, causing a local perforation not necessarily connected with the lumen of the appendix; (2) in detachment of the appendix, if the necrosis involves the whole circumference; or (3) in complete gangrene of the whole organ. It is due to micro-organisms, among

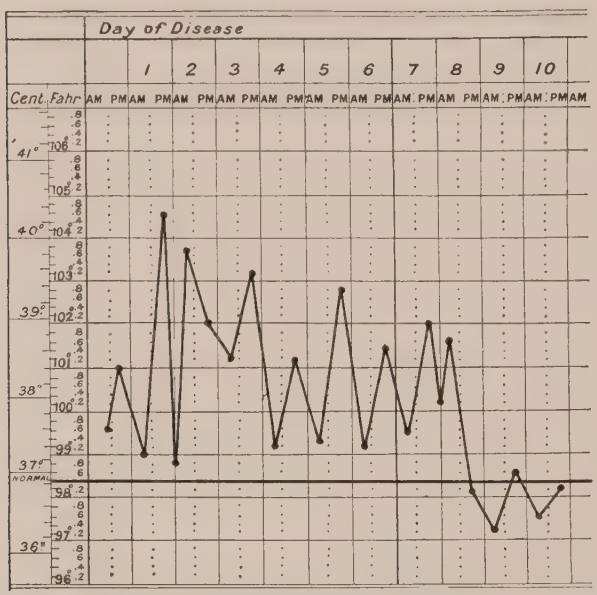
[1] "Diseases of the Vermiform Appendix," London, 1895.

which the bacillus coli communis is that most often found, though it seems probable that in the initial stage at least the active agent is one of the pyogenic streptococci. The importance of inflammation of the appendix lies entirely in the risk of the production of peritonitis. Obviously this risk varies with the nature of the primary lesion. Thus chronic catarrh or stricture with cystic dilatation gives rise most commonly to local adhesive peritonitis: acute inflammatory necrosis, and ulceration due to concretion, may cause either acute local peritonitis with formation of pus (perityphlitic abscess) or acute general peritonitis.

In cases of **local adhesive peritonitis** there is, as a rule, no discoverable determining cause, but occasionally the onset is preceded by a meal of indigestible food, or by a blow or strain. Pain comes on rapidly, sometimes so suddenly that the child cries out as though struck. It is referred, as a rule, to the right iliac fossa, but may at first be diffused over the abdomen, and localized only on the second day. It radiates towards the umbilicus, and may extend to the front of the thigh in the region supplied by the anterior crural nerve. Vomiting may occur once or twice at the onset, and constipation due to paralysis of the gut is the rule from an early stage, though two or three motions may be passed at the onset. The temperature rises at once, and commonly attains its maximum on the first day. It is accompanied by a good deal of general depression, which may even amount to collapse. The pulse is quickened in proportion to the fever. The tongue is furred but moist, and there is anorexia. The right thigh is flexed upon the abdomen, and attempts to extend it cause severe pain. With rest the pain subsides in the course of a few days, but marked local tenderness persists somewhat longer. It is usually most marked at McBurney's point (about halfway between the anterior superior iliac spine and the umbilicus). It may extend over the whole of the right lower quadrant of the abdomen and as high as the ribs. At first palpation discloses no more than increased resistance in this area, due to rigid contraction of the abdominal muscles, but as the acute process subsides a soft, ill-defined swelling may usually be felt, generally oval in form, with its long diameter parallel with the outer part of Poupart's ligament. Should the appendix occupy one of the less usual positions mentioned above, the area of tenderness and the swelling will be displaced correspondingly. The tumor is due to congestion and swelling of the cæcum and the lower part of the ileum, with fibrinous exudation between the coils; its bulk is frequently increased by some fæcal accumulation. Resonance over the swelling is generally but not invariably diminished. In a simple attack of this nature the temperature usually falls to normal about the seventh day, and the patient is convalescent in ten days or a fortnight, but similar attacks may recur at more or less frequent intervals.

Perityphlitic Abscess.—The early symptoms in a case in which suppuration eventually occurs are identical with those just described, and may not be exceptionally severe. Suppuration may begin as early as the fourth day, but this is unusual; its occurrence is indicated by the rapid formation of a large tumor, harder and better defined than in simple adhesive peritonitis. In less acute cases the persistence, or even the increase in size of the swelling after a week or ten days have passed, and the increase of tenderness will suggest the presence of suppuration. The temperature is also of assistance since when suppuration occurs the normal fall on the sixth or seventh day

FIG. 54.



Temperature chart of a case of simple Appendicular Peritonitis in a child aged 14. (Hawkins "Diseases of the Vermiform Appendix.")

does not take place, or it is interrupted by a secondary rise which presents the morning remissions and evening exacerbations commonly produced by suppuration. Examination under chloroform, which in doubtful cases should never be omitted, may reveal fluctuation, and render a positive definite diagnosis possible. Apart from fluctuation, to the absence of which not much weight can be attached, the most reliable signs of suppuration are the character of the temperature and the increased tenderness, since a swelling persisting for several weeks may be due to a mass of thickened adherent omentum. In rare cases only will the continuous temperature be due to one of the complica-

tions of perityphlitic abscess, such as infective thrombosis of the portal vein and hepatic abscess. Perityphlitic abscess may rupture into the gut, or it may point at the surface, leading in some instances to fæcal fistula. Inflammation of the psoas muscle may occur, and may account for long-persistent flexion of the hip. The most serious complication is intestinal obstruction due to kinking of the small intestine, produced by adhesion of one of its coils to the inflammatory mass in the left iliac fossa.

Acute general peritonitis due to inflammation of the appendix generally comes on quite suddenly in the midst of apparent health, and in a child who has never presented any symptoms referable to the appendix. In some cases the history renders it probable that peritonitis, which might have been localized, is made general by the administration of violent purgatives as soon as complaint was made of pain in the abdomen. The symptoms do not differ from those of general peritonitis from other causes—general abdominal pain and acute tenderness and distension, followed within twenty-four hours by arrest of the abdominal respiratory movements. The pain is not localized, and its onset is followed quickly by vomiting. The mouth becomes dry, thirst is extreme, and the tongue is furred; the urine is scanty, and often contains albumen; the pulse is quick; the temperature rises rapidly to 102° or 103°, but usually falls before death; the face is pinched and anxious, and the patient retains consciousness until shortly before death, when anxiety is replaced by apathy and somnolence. Both legs are drawn up, and in the less acute cases evidence of fluid in the abdomen may be discovered.

The **diagnosis** of localized peritonitis due to inflammation of the appendix is not, as a rule, difficult. From intussusception it is distinguished by the fact that in this condition the tenderness and signs of local peritonitis are not early symptoms; that the tumor, if discoverable, is found in the middle line or towards the left, and seldom occupies the right iliac fossa, where the sense of resistance is diminished, or at least not increased; and that tenesmus is an early and prominent symptom. Fæcal accumulations may produce pain, vomiting, constipation, and slight fever. The constipation may be replaced by diarrhœa, or at least by the passage of frequent small stools, but the diagnosis in the early stage and on a single examination is often difficult. The pain is colicky and intermittent, and in place of muscular rigidity in the right iliac fossa there is in this or some other region a distinct tumor, which is not tender, though colic may be determined by its manipulation. [A blood count is here of great value, and leucocytosis, if it be found, points strongly towards appendicitis especially if several different counts show an increasing number of whites. In the most severe cases of appendicitis, however, there may be no leucocytosis, absence of which

in such cases, is a grave prognostic sign.] Probably the condition with which appendicular peritonitis is most often confounded is disease of the right hip joint, which it resembles owing to the flexion of the hip, the limp in walking, and the pain produced by handling the limb. It will be found, however, in appendicular disease that though the thigh cannot be extended without causing acute pain, it can be rotated without complaint; that there is no tenderness behind the trochanter, and no wasting of the muscles of the thigh or displacement of the gluteal fold; and that the child while lying down will spontaneously increase the flexion of the joint or permit this to be done, without giving any signs of suffering. Abscess in the iliac fossa from other causes must be borne in mind. Among these may be enumerated caries of the spine or of the innominate bone, disease of the sacro-iliac joint, empyema tracking downwards, and superficial abscess, the result of injury; perinephritic abscess, though a very rare condition in children, may also be mentioned. The history, which in the majority of the conditions enumerated is prolonged, in contrast with the sudden onset of appendicular peritonitis, will assist in the diagnosis, which, however, can usually be made with certainty only after careful physical examination under chloroform.

The **treatment** of local peritonitis due to appendicular disease should consist of rest in bed, with a pillow under the knee on the affected side, the application of poultices or fomentations, or of an ice bag to the right iliac fossa to relieve pain, and the administration of opium (♏ij every four hours to a child of five) or of opium and belladonna if vomiting is troublesome. A hypodermic injection of morphine (gr. $\frac{1}{16}$) may be given, but in either case it is unnecessary, and certainly undesirable, to continue the use of opiates for more than two days. No purgative or laxative medicine should be given but if it appears desirable to solicit an action of the bowels, and this should not be done until pain has subsided and convalescence is commencing, a glycerine suppository or a small glycerine enema (℥j to ℥ij), or a simple soap enema, should be given. The management of convalescence is of great importance. Absolute rest in bed is essential, but though it is easy to keep a child in bed, it is difficult to keep a robust boy at rest after the local pain and tenderness have subsided. For this reason it has seemed to me advisable to apply a long splint for a week or a fortnight. During convalescence purgatives should be avoided, and at most a mild saline aperient should be given if an enema is deemed insufficient. When there is evidence of suppuration, or when there is reason to suspect it, and the patient's general condition is deteriorating, surgical interference should not be delayed. While the temperature is elevated, and until convalescence is completely established, the diet should be fluid, and in the early stage at least pancreatized milk, or some other form of

predigested food, is to be preferred. The treatment of general peritonitis must be directed to maintaining the strength and relieving pain. The use of saline laxatives, which has found much favor in peritonitis due to other causes, is contraindicated, and the too free administration of opium masks the symptoms. In 9 of the 36 cases observed by Hawkins recovery ensued. In 11 cases the abdomen was opened, the pus and inflammatory products within reach of the incision removed, and an attempt made to wash out the whole abdominal cavity; none of these cases recovered, so that this, which seems to be the most rational treatment when the diagnosis of appendicular disease can be made with an approach to certainty, should only be adopted after the most careful consideration.

CHAPTER XXX.

DISEASES OF THE LIVER.

Jaundice—Catarrhal Jaundice—Infective Jaundice—Acute Yellow Atrophy—Cirrhosis—Amyloid Degeneration—Fatty Infiltration—Fatty Degeneration—Suppurative Hepatitis.

Pathological and clinical conceptions as to diseases of the liver are so incomplete and so governed by tradition that it is difficult to arrive at a satisfactory classification. The liver, like other glandular organs, consists of a secreting epithelium, excretory ducts, and blood-vessels, though the blood supply presents certain well-known peculiarities related to the assimilative functions of the gland. Any one of these parts may be the primary seat of morbid changes of inflammatory or degenerative type. Thus there may be catarrhal inflammation of the bile ducts causing catarrhal jaundice, purulent inflammation leading to suppuration and abscess of the liver, or fibrosis producing so-called biliary cirrhosis. The glandular substance itself is liable to acute degeneration, probably of the nature of infective inflammation, of which acute yellow atrophy is the best known and most marked form; to fatty degeneration; to fatty infiltration; and to atrophy by compression produced by fibrous overgrowth. The blood-vessels are liable to infective inflammation (pylephlebitis), producing disseminated abscesses, and to fibrous overgrowth of their connective tissue sheaths leading to cirrhosis.

The double function of the liver as an assimilative and a secreting organ is disturbed to a greater or lesser extent by affection of any of its parts, but most profoundly by affections of the hepatic cells. Thus we have on the one hand imperfect assimilation, especially of fats and carbohydrates, and on the other imperfect formation of bile, or retention and absorption, jaundice being in either case produced.

Jaundice is due to absorption in the liver of bile pigment which is carried by the blood to all the organs and tissues of the body. Bile salts and certain toxins are also absorbed. Bilirubin is itself toxic, producing extreme slowing of the heart and a fall of blood pressure. Bile salts have a similar but less marked action. Bilirubin is the pigment absorbed in ordinary cases of catarrhal jaundice. When the hepatic glandular substance is affected primarily, its production of pigment is imperfect and urobilin is formed, absorbed,

and excreted in the urine. This abnormality in the functions of the hepatic cells is attended by other changes in the constitution of the bile, which favor the absorption of bile pigments independently of obstruction of the bile ducts. Among these should be mentioned a thickening of the bile, which causes it to flow less easily.

When the functions of the liver are deranged suddenly there is a disturbance of nitrogenous metabolism, and a diminution in the excretion of urea by the urine, except in extreme cases (acute yellow atrophy), when there is at first an excess of urea, together with the appearance of products of imperfect metabolism (leucin, tyrosin, xanthine, etc.), which subsequently replace urea almost entirely. With the restoration of the functions of the liver there is a large increase in the urea, in the bulk of the urine, and in the proportion of toxins which it contains. Bile salts are seldom present in the urine, except in the terminal stage of acute yellow atrophy. The extent to which the assimilative functions may be deranged is shown by the fact that in some cases there may be temporary glycosuria attendant upon the ingestion of carbohydrates (alimentary glycosuria).

The liver also is liable to become infected by tuberculosis and syphilis, to be the seat of hydatids (see page 407) and of new growths, though these are of very rare occurrence in childhood. It may be useful to insert here the following classification of morbid processes to which the liver is liable, though it will not be convenient to follow the arrangement closely in the following pages :—

Affections of bile channels.....	Catarrhal jaundice.
	Infective catarrhal jaundice.
	Purulent Inflammation, abscess.
	Biliary cirrhosis.
Affections of hepatic cells.....	Infective jaundice.
	Acute yellow atrophy.
Affections of blood-vessels.....	Cirrhosis.
	Pylephlebitis.
Syphilis.	
Tuberculosis.	
Hydatid disease.	
New growths.	

Catarrhal jaundice is by no means uncommon in children; it may occur in infancy, but is comparatively rare under three years. Occasionally it prevails in an epidemic manner.

The majority of cases of simple jaundice, in children as in adults, are due no doubt to catarrhal inflammation, more or less severe and extensive, of the bile duct and possibly of its tributaries. This angiocholitis, which is usually associated with duodenal catarrh, and preceded by gastric catarrh, produces a thickening of the mucous lining, which becomes injected and gelatinous, and secretes an abnormal amount of tenacious mucus. Among the causes predisposing

to catarrhal jaundice must be reckoned errors in diet, excessive quantities of fatty and nitrogenous foods, and alcohol, which is by some parents given in considerable quantities at a very early age. In other cases exposure to emanations from foul drains or from collections of decomposing animal matters, or bathing in sewage-polluted rivers, has appeared to be the determining cause of the jaundice. The cause of an epidemic of jaundice has usually been found in some such conditions as those just enumerated. In some cases, of which those due to the causes last mentioned perhaps constitute the majority, the jaundice is due to a progressive infection, which involves not only the bile vessels but also the hepatic cells. This affection, though graver than ordinary catarrhal jaundice from retention, presents symptoms which are of the same kind but more marked, especially at the onset.

The characteristic symptoms of catarrhal jaundice are preceded usually for three or four days by malaise, headache, loss of appetite, gastric uneasiness, and nausea, which may lead to vomiting. The tongue is large, covered with a thick white fur, and the breath is very offensive. These symptoms may have passed away, and the tongue may have become clean before the icteric tint is noticeable. The characteristic golden-yellow staining of the integumentary structures will be seen first over the sclerotics, then on the lips, the hard palate, the corners of the mouth, the temples and forehead ; a little later it becomes evident over the trunk, and last on the extremities. Some twenty-four hours before any pigmentation can be noticed the urine will have contained bile pigments, which impart to it a color varying from a greenish-yellow to a dark brown. Both the quantity of urine and urea is diminished. The pigment is taken up by the cells of the Malpighian layer of the skin, and retained by them probably until they are exfoliated, so that the yellow coloration persists for two or three weeks after its cause has been removed. All the organs of the body, with the exception of the central nervous system, are bile-stained. The liver is a little enlarged, tender on firm pressure, and soft. The fæces, which are usually pasty, are of a light color, so that they are compared to moist clay. The want of color is due in part to the absence of bile pigment, and in part to the presence of an excessive quantity of fat which, in the absence of the bile from the intestine, is imperfectly absorbed. More than half the fat ingested may be eliminated with the fæces. The stools have an offensive odor of putrefaction, and the aromatic sulphates of the urine, which vary directly with the amount of intestinal putrefaction, are increased. The slowing of the pulse, which is a marked and constant symptom in adults, is, as Henoch has observed, not often noticeable in young children, owing perhaps to the nervous excitement which a medical examination causes in them. Pruritus also,

which is frequently the most distressing symptom in adults, is often absent in children. Of the subjective symptoms, the most marked are mental depression, heaviness, and drowsiness. Complaint is often made of a sense of fulness in the hepatic region, of a bitter taste in the mouth, and in a few rare cases xanthopsia has been definitely present, owing apparently to affection of the cerebral centres. On the whole, however, the symptoms, both those which precede and those which accompany the jaundice, are slight, and medical aid is often sought only when the yellow tint has become well established, and the patient is, in fact, already convalescent.

Infective Jaundice.[1]—The presence or absence of fever is a point of much interest and importance. In the greater number of cases no elevation of temperature occurs, or it is very slight and of short duration. In others there is very well-marked pyrexia in the early stage. Such cases are of a more severe type in other respects; they are met with most often during epidemics; and there is probably some involvement of the hepatic glandular tissue in the infective process. A day or two after the exposure to the supposed cause, or even after a shorter interval, the patient begins to suffer from aching pains in the joints and back; from depression, loss of appetite, nausea or vomiting, giddiness, and a bitter taste. The temperature is found to be raised, and ranges for some days between 101° and 103° F. The urine is scanty and high-colored, the spleen is enlarged, the liver enlarged and tender, epistaxis sometimes occurs, and there is often an outbreak of herpes labialis. After five or six days jaundice appears, the fever abates, large quantities of urine containing much urea are passed, and the general condition improves rapidly. At or about the time at which the jaundice appears the fæces becomes clay-colored, but, as a rule, convalescence is not interrupted.

There is another variety of infectious jaundice to which Chauffard[2] proposes to apply the term "*pleiochromic.*" It is of the same type as the form last mentioned, but more severe. There is a primary affection of the hepatic glandular tissue, causing an alteration in the chemical constitution of the bile, and an excess of coloring matter which is absorbed and produces jaundice, though there is no retention, and the fæces are dark-colored. The patient is taken ill suddenly with headache, aching in the limbs and back, fever, loss of appetite, nausea, often vomiting, and enlargement of the spleen and albuminuria. There may be slight wandering delirium, and the general resemblance to typhoid fever may be very close. On the fifth or sixth day jaundice appears, accompanied often by petechiæ,

[1] Dr. William Hunter's article in Prof. Clifford Allbutt's "System of Medicine," published while these pages were passing through the press, deals with this and cognate subjects in a masterly manner.

[2] "Traité de Méd." (Charcot, Bouchard, Bouillaud), p. 754.

by epistaxis, and by bilious diarrhœa. Two or three days later the temperature falls; a large quantity of urea is eliminated in the urine, which becomes copious and free from bile pigment and from albumen. The fæces becomes normal, and the patient recovers slowly. In some cases convalescence is interrupted in a week or less by a relapse. Herpes of the lips has been observed, and a roseolar or scarlatiniform rash has been met with during the pyrexial period, and severe urticaria at its close. This is a picture of an infective disease, and Ducamp states that it has an incubation period of five days. The resemblance of the symptoms of the early stage to typhoid fever has been mentioned, and the disease has been supposed to be due to a special localization of the typhoid bacillus in the liver and bile channels. Direct evidence on this head is wanting. Such as exists is, on the whole, opposed to the theory, although there are good grounds for believing that the infective agent is derived from water or air contaminated by sewage or the products of the putrefaction of animal matters. Chauffard's view that the "new infectious disease" described by Weil, and known as Weil's Disease, is identical with this form of jaundice appears to be well grounded. The severe forms of icterus neonatorum (*q. v.*) associated with septic infection belong to the same class, the jaundice being due to an infective lesion of the hepatic cells.

As the final term of this series of hepatic disorders we have *acute yellow atrophy of the liver*, in which there is a rapid granular degeneration of the hepatic cells, and consequent shrinking of the whole organ. There is coincident glomerulo-nephritis and enlargement of the spleen. The onset may be sudden or insidious, and the symptoms resemble those of the form last described, but are more intense. The fæces after a time lose their bilious color, owing to the arrest of the secretion of bile due to the progressive destruction of the hepatic glandular tissue. There is pain and tenderness in the hepatic region, and rapid diminution in the area of hepatic dulness. The temperature, elevated at first, tends to fall after the first six or seven days, while the pulse which is small and soft, becomes progressively more rapid. The urine is scanty, and contains at first an excess of urea, but subsequently urea almost disappears, its place being taken by leucin, tyrosin, etc. The blood, which is dark and does not coagulate readily, also contains large quantities of these products of imperfect nitrogenous metabolism. The disease, which is extremely rare in childhood, is due probably to an infective agent derived from insanitary surroundings; among predisposing causes are alcoholism and syphilis.

The **prognosis** of simple catarrhal jaundice is good, and in the more severe forms it is not unfavorable, especially in children, in whom the alcoholic habit is seldom confirmed. Previous disease of the

kidneys is an unfavorable element in prognosis, since the danger to life lies in the retention of toxins which are produced owing to the deficient functional activity of the liver; when the kidneys are healthy these are eliminated rapidly. The prognosis is less favorable when there is well-marked fever and when the stools are bilious, but recovery is the rule, except in acute yellow atrophy, which is a very fatal disease.

The **treatment** of simple catarrhal jaundice is directed to the removal of the obstruction, and to the prevention of intestinal putrefaction and the absorption of toxic production. Calomel in small doses, frequently repeated, is a valuable drug, since it not only stimulates peristalsis but exercises an antiseptic action, owing probably to the perchloride of mercury which it contains in small quantities. Salol, naphthol, benzonaphthol, and salicylate of bismuth also are useful to correct the putrefaction of the intestinal contents. Chauffard recommends a combination of salicylate of naphthol with salol, since these drugs are not only intestinal antiseptics but also cause the appearance in the bile of salicylic derivatives, which have an antiseptic action on the bile channels. Later, when the fæces begin again to be of natural color, a mixture containing hydrochloric or nitro-hydrochloric acid should be given, and a small dose of liquor strychninæ or tincture of nux vomica is a useful addition. The use of rhubarb in various combinations, and of senna, which was very popular, appears to offer no advantages which compensate for the nauseous flavor of these drugs, and in a condition such as this, when the object should be to promote appetite and improve digestion, prescriptions cannot be too simple. The food should be very simple, and the greatest advantage is to be derived from a diet consisting exclusively of milk or skim milk and whey. It is sufficient and simple, while it has a diuretic action which is desirable. As beverages, freshly-made lemonade, lemonade made with barley-water, or the Imperial drink may be permitted, but all alcoholic drinks should be forbidden. Meat should not be permitted, and the use of broths and beef-tea is better avoided. The patient should at first be kept at rest, and confined to bed should there be any fever. Later, regular exercise should be prescribed, and should the liver continue tender or a tendency to constipation remain, a course of saline laxatives, either at home or at a spa, should be prescribed.

In the graver forms of jaundice, and in acute yellow atrophy, the indications for treatment are the same. Owing to the fact that the disease is attended by deficient oxidation of nitrogenous bodies (elimination of leucin, tyrosin, etc.), inhalation of oxygen and the internal administration of drugs believed to favor oxidation, such as benzoate of soda, have been recommended.

Cirrhosis of the liver is a rare disease in childhood. Alcoholism,

the commonest single cause, accounts for about one-sixth of the cases. The production of fibrosis of the liver by syphilis and by tuberculosis accounts for about one-fifth of the cases. Of the remainder the major part occur as sequelæ of acute infectious diseases, especially scarlet fever and measles.

Most of the cases are examples of portal fibrosis, but the liver is more often large (hypertrophic) than contracted. The organ is large and heavy, its edge blunt, its color a gray or reddish yellow, its cut surface firm and finely granular. The large size of the liver is due mainly to the fact that the hepatic cells do not atrophy, nor does the fibrous growth retract.

The prominent symptom, and that first noticed, is ascites; the limbs are wasted, the skin has a waxy tint, and œdema of the lower extremities may be an early symptom. The subcutaneous veins of the abdomen may become much dilated, forming four or five large trunks, which run down from the neighborhood of the xiphoid cartilage to the groin. They communicate above with the epigastric and internal mammary, and below with the iliac and saphenous veins. The course of the blood in them is from above downwards. This dilatation commences later than ascites, and may persist long after it has ceased; it is certain evidence of portal obstruction. The enlarged liver may be felt unless the ascites is extreme, and the spleen also is, as a rule, enlarged. The quantity of urine passed is small and the urea below, but the uric acid above, the normal. The nose bleeds easily, but epistaxis is not severe, whereas the patient is liable to severe gastro-intestinal hæmorrhage. The stools are not pale, and diarrhœa is common. Chronic peritonitis may be associated with cirrhosis.

The **prognosis** of hypertrophic fibrosis is fairly good if the case can be treated systematically from an early stage.

The **treatment** must be directed in the first place to the removal of the cause, and in particular all alcoholic stimulants should be arrested. The patient should be put on a diet of milk, with skim milk or whey as a beverage, and the diuresis to which this diet predisposes encouraged by diuretic drugs, of which the most valuable is calomel. It not only exerts a marked diuretic action but stimulates the hepatic cells, and possibly also tends to arrest the fibrosis. It should be given in small doses, gr. $\frac{1}{4}$–$\frac{1}{2}$ daily. During the course of calomel antiseptic mouth washes should be used, and the teeth kept scrupulously clean. Potassium iodide has been used, but it is of doubtful value, and its effect must be carefully watched. Diarrhœa should not be checked unless very copious. If the amount of fluid in the peritoneum is large, it must be withdrawn by tapping, and the operation must be repeated when necessary.

Hypertrophic biliary cirrhosis, due to fibroid proliferation in connection with the bile vessels, and characterized by enlargement of

the liver and repeated attacks of jaundice, has been observed in childhood, but the number of recorded cases is very small. The liver is generally tender, there is no ascites, the fæces are soft, and of a gray, brown, or light yellow color. The spleen is enlarged. Hæmorrhages from the nose and intestines are frequent and often severe. The urine contains bile pigment. The disease is progressive. The patient becomes exceedingly weak, and succumbs in one of the attacks of jaundice. The treatment which appears to offer the best hope is a milk diet, systematic use of intestinal antiseptics, and of minute doses of calomel.

Amyloid degeneration of the liver is associated with syphilis, or with long-continued suppuration of pulmonary cavities, of the pleura, of joints, bones, or glands (chronic tuberculosis). The improvement which has taken place in surgical methods has rendered it less common than formerly. The degeneration begins in connection with the capillaries and arterioles, which become enveloped in a sheath of homogeneous transparent material. This fact lends support to the theory, which the etiology suggests, that the degeneration is due to some peculiarity of the blood, possibly to the presence in it of some toxin derived from the suppurating lesion. Later, the degeneration extends to the liver cells. The amyloid substance is a nitrogenous body and is very little subject to change. It is insoluble in the gastric juice, in acids, and in alkalies, and it does not easily undergo decomposition by putrefaction. Treated with a weak solution of iodine it takes a dark walnut color, which is changed to blue, and finally to purple by sulphuric acid. The liver when affected by amyloid degeneration is large, firm, and painless, with rounded edge. The spleen is usually enlarged by a similar amyloid degeneration, which may affect other organs also, particularly the intestinal mucous membrane. The patient makes no complaint with reference to the liver, and the condition is usually only discovered on physical examination, for which an indication is afforded by a deterioration in the nutrition, and by clubbing of the fingers.

Amyloid degeneration is not in itself susceptible of treatment. If its cause can be removed the patient rapidly improves, but from the nature of the determining causes such patients are seldom long-lived, though the part which the amyloid degeneration has in bringing about this result, and indeed its fate subsequent to the arrest of the suppuration, is not well understood. The occurrence of this degeneration certainly renders the prognosis worse in those cases of joint or lung disease in which it occurs.

The enlargement of the liver, so often present in *rickets*, has been attributed to amyloid degeneration, but in the majority of cases at least it is due to **fatty infiltration** of the liver cells, which causes a general enlargement of the organ. The substance of the liver is of

a lighter color and softer consistency than natural. The quantity of fatty matter in such a liver may be increased to six and even ten times the normal. This condition, in which there is merely an infiltration of fat to a large extent taking the place of the water of the liver, and in which there is not, in fact, any structural lesion, must be distinguished from **fatty degeneration**, which is a consequence of the granular degeneration, or cloudy swelling, which occurs in the majority of acute febrile diseases, including acute pulmonary phthisis. In this condition the fat is derived from a degeneration of the glandular protoplasm, and a similar degeneration may attack the epithelium of the blood-vessels and capillaries.

Suppurative hepatitis is uncommon in children. It may be secondary to suppuration in connection with the appendix vermiformis, or of the mesenteric glands in typhoid fever, to dysentery, or to pyæmia. In a few instances it has been caused by the entrance of a round worm into the bile passage. With this exception, indeed, its causes are the same as in adults, and its symptoms and course are identical.

CHAPTER XXXI.

ACUTE DISORDERS OF THE GASTRO-INTESTINAL SYSTEM.

Etiology—Dyspepsia—Catarrhal Enteritis—Gastro-intestinal Catarrh—Acute Gastro-enteritis—Acute Summer Diarrhœa—[Bacteriology]—Cholera Infantum—Complications—Treatment.

The **gastro-intestinal mucous membrane** is the largest gland in the body. In its pathological relations it presents certain analogies to the skin, since it is liable to be exposed to the direct action of irritating substances, and is constantly in relation with numerous bacteria. Some are harmless, perhaps even useful in digestion, though the *bacillus coli communis* and, possibly, others may under certain circumstances become pathogenic. Further, the food may contain microbes essentially pathogenic, as, for instance, the bacillus of tubercle, and that of typhoid fever. Poisonous substances may be introduced into the canal with food or drink, or may be produced within it by perversion of the process of digestion. These poisons acting on the mucous membrane may cause local irritation and disturbance of function, or catarrh, or they may be absorbed, and so produce general symptoms. In the latter alternative local lesions may not be produced, or they may be slight or late in making their appearance. Thus in many cases of even serious general disturbance traceable to the gastro-intestinal canal, the morbid changes in the mucous membrane are recognized with difficulty, if at all, and may be, and probably are, of secondary importance. The most serious symptoms of such disorders are secondary not to lesions of the mucous membrane but to the absorption of poisonous substances introduced into the alimentary canal, or produced within it.

Gastro-intestinal disorders may also be *secondary* to general diseases, especially the acute infectious diseases, as, for instance, measles, and are then produced either by the action of the specific infection on the mucous membrane, or by the elimination of toxic bodies through it. It may be the site of specific infections, as in enteric fever and tuberculosis, of new growths, or of parasites. Disturbance of its functions may be due to derangement of the nervous mechanism. Malformations and malpositions, congenital or acquired, may under certain circumstances give rise to serious symptoms.

At the present time it is not possible to make a rational *classification* of gastro-intestinal disorders, founded either upon morbid anatomy or on the nature of the bacterial infection which undoubtedly has a large share in producing diseases having their origin in this tract. The attempt to found a classification on the presence or absence of inflammation is not successful because, in the first place, the question whether inflammatory changes are to be observed depends in many cases, if not in the majority, on the duration of the disease, such changes making their appearance if the patient survive the severe general symptoms produced by the absorption of toxic bodies, which are the true *materies morbi;* and because, in the second place, it may be difficult to decide whether certain slight changes observed in more chronic cases ought to be regarded as inflammatory or degenerative.[1] On the whole, it will be found most advantageous to follow clinical features, which are, as a rule, related to certain etiological factors to be more or less clearly discerned.

The *diet* may be imperfect in more than one way. Thus (1) there may be deficiency or excess in the amount of food given, or in the proportion in which certain constituents are present in it. For example, cow's milk diluted to reduce the quantity of albumen to that present in human milk contains too little fat and milk-sugar, and the diet of "tea" and bread-and-butter, on which too many children are fed immediately after weaning, is deficient in proteid and in fat.

(2) The food may have undergone fermentative changes. Sour milk, milk "on the turn," meat which has undergone putrefactive and other allied changes, and bad fruit come under this category.

(3) The food given, though of good quality, may be unsuited to the digestive powers of an infant or child newly weaned; for example, solid meat, green vegetables, potatoes, and the ordinary food of the table. Such a diet is, moreover, usually deficient in certain constituents, especially fat.

In *infants* at the breast **over-feeding** is the most common cause of dyspepsia. Too frequent suckling is the most common cause, but in exceptional cases the milk is itself too rich. The infant suffers from colic and flatulent distension; it lies on its side, with its legs drawn up. It cries, and is suckled again to "pacify it." The bowels are at first constipated, but, later, a motion is passed usually after each suckling. This condition may usually be relieved by giving one or two doses of castor oil or liquorice powder, or calomel (gr. ss) to unload the bowels, and by directing that the infant shall be suckled every three hours only. If the milk be too rich a little boiled water, about ℥ss, sweetened with milk-sugar should be given before each suckling.

[1] Another and very real difficulty lies in the danger of mistaking *post-mortem* for pathological changes.

If the **dyspepsia** persist, a change is observed in the color of the stools, which lose their golden-yellow color, and become green. In the mildest cases the change in color takes place only after the stools have been passed; in the more severe it occurs within the intestine, but seldom higher up than the middle of the jejunum. It is due to the oxidation of bilirubin to biliverdin, and is to be attributed, probably, to some alteration in the activity of the secreting organs, and not to the action of bacteria. The green color may cease spontaneously in a day or so, and may, indeed, recur several times before any other symptoms appear. It may occur before there is any evidence of gastric irritation. As a rule, it is accompanied by some increase in the number of the motions, which are more watery than in health. This is due to increased peristalsis, which hurries the fæcal matter through the large intestine, where under normal circumstances it undergoes inspissation. These green stools, are commonly very acid, and often produce excoriation and intertrigo of the anus and buttocks.

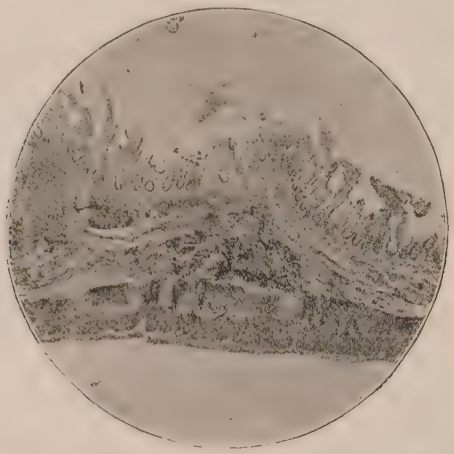
If the cause of the disorder be not removed, the stools next become slimy owing to the secretion of an excessive quantity of mucus derived especially from the large intestine. At the same time, a considerable formation of gas produces distension and discomfort. It is passed often with the motions, which are in consequence expelled with explosive violence. These symptoms are attributed to catarrh, and the condition is often spoken of as **catarrhal enteritis**. Peristalsis becomes irregular and painful, and the infant is restless, kicks and struggles, or keeps the thighs drawn up to the belly. The attacks of painful colic cause sudden outcries, "screaming fits," and long periods of continued crying and whining. Symptoms of gastric irritation may supervene at any time. Appetite is diminished, and the infant, after suckling eagerly for a minute or two, ceases and begins to cry owing to the painful colic which has been excited by the ingestion of the milk. The milk may be vomited immediately, or at a later hour curds may be brought up, often in large masses, sometimes of a yellow color and mixed or coated with mucus. The normal increase of weight is checked at an early stage, then a loss of weight begins, and eventually extreme emaciation may ensue.

The *morbid changes* to be observed in the intestines, in addition to the increased secretion of mucus, are that the superficial parts of the mucous membrane are, as it were, infiltrated with mucus, and that there is a considerable shedding of epithelial cells, which are entangled in the mucus with which the surface is covered. The contents of the small intestine are watery. The stools contain a large proportion of nitrogenous principles, though the actual daily loss of nitrogen by the bowels is not greatly increased.

Green over-liquid stools should be *treated* by alkalis to relieve

symptoms, and careful investigation should be made as to any errors in the suckling and clothing of the infant. The alkali used may be lime-water or sodium carbonate, or the old-fashioned powders of

FIG. 55.



Catarrhal swelling of the intestinal mucous membrane, with partial destruction and detachment of superficial epithelial layers (early stage). (× 30.)

rhubarb with soda or magnesia (*e. g.*, Pulv. Rhei Co., gr. iij–v, t. d.). If the stools are slimy when the infant comes under treatment, it should be given a drachm of castor-oil to clear out the intestines,

FIG. 56.

Localized destruction of superficial layers of mucous membrane (later stage). (× 32.) (Photomicrographs by Professor Baginsky.)

and subsequently for a few days small doses of the same drug combined with an alkali, as in the ordinary castor-oil mixture. In some cases these may be replaced with advantage by small doses of magnesium sulphate.

The severer forms of dyspepsia and chronic catarrh are comparatively rare in infants reared at the breast, but in those brought up by hand dyspepsia is apt to become a serious disorder. It arises more frequently, its symptoms are more severe and less amenable to treatment, they lead to structural changes in the intestinal mucous membrane, and predispose to acute febrile diarrhœa and cholera infantum. The early stages may be the same as those already described in suckling infants, but the later stages of the disorder differ in nature and seat.

In some cases symptoms referable mainly to the small intestine persist, and the infant gradually grows weaker. In others gastric symptoms become more prominent, and the strength is reduced more rapidly by frequent vomiting and complete anorexia. In others, again, the most pronounced symptoms are those of colitis.

The course of the severer forms of **gastro-intestinal catarrh** is very inconstant. In the early stage arrest of the symptoms is followed by rapid improvement, but relapses are very apt to occur. In the later stages recovery is much more slow owing to impairment of digestion, and the liability to relapse is greater. Fever may be absent even down to a fatal termination, but in other cases there is with each relapse or exacerbation a rise of temperature which lasts for a few hours or days. In others, again, especially those in which the symptoms point most distinctly to catarrh of the small intestine, remittent fever may persist for weeks, the thermometer rising in the evening to 100° or 101° F. When exhaustion is great, however, the temperature is usually subnormal and may fall to 95° or 96° F. In the most acute cases the infant becomes prostrate rapidly, and may die within two or three days; in the mildest, convalescence is established within five or six days; in the majority, however, the acute is followed by a subacute or chronic gastro-enteritis, which may last for weeks, months, or years.

Acute gastro-enteritis may occur at any age, but is met with perhaps most often in children two or three years old who have suffered since infancy from repeated attacks of gastro-intestinal catarrh. Not infrequently, however, it comes on acutely in a child who has previously enjoyed fairly good health. Such attacks may be traced to chill, owing to insufficient clothing of the abdomen and lower limbs, or they occur during the summer months under conditions similar to those which produce cholera infantum. More rarely the condition is a complication or sequela of one of the acute infectious diseases, especially measles.

There is a general catarrhal inflammation of the ileum and colon, with shedding of the epithelium (Fig. 55) and infiltration into the mucous and submucous tissue. The cells of Lieberkühn's crypts also undergo disintegration (Fig. 57), and finally there is a local destruction (epithelial necrosis) of the superficial layer of the mucous membrane (Fig. 56). Changes of a similar kind, but less severe, take place also as a rule in the mucous membrane of the stomach. In other cases the inflammatory process is from the commencement less superficial; the mucous membrane is swollen owing to the infiltration of the submucosa with round cells (Fig. 58). The

FIG. 57.

Acute catarrhal enteritis. Disintegration of the epithelium of Lieberkühn's crypts. (× 100.) (Photomicrograph by Professor Baginsky.)

lymph follicles are the parts most affected, and the projection of the distended follicles (Fig. 60) gives to the mucous membrane a granular appearance. Finally the follicle breaks down and discharges through an aperture formed through the superficial layers of the mucous membrane, and a small circular ulcer results (Fig. 59). To this form the term follicular enteritis is applied.

The onset of acute gastro-enteritis, when sudden, is often attended by vomiting, the temperature rises, and the child becomes restless and often perspires freely. It then has two or three motions consisting of fæcal masses suspended in a brownish fluid. After a short

FIG. 58.

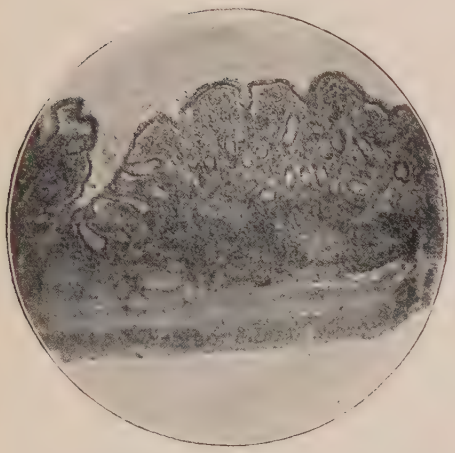
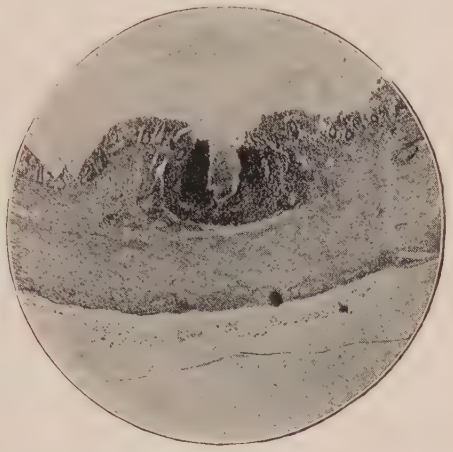
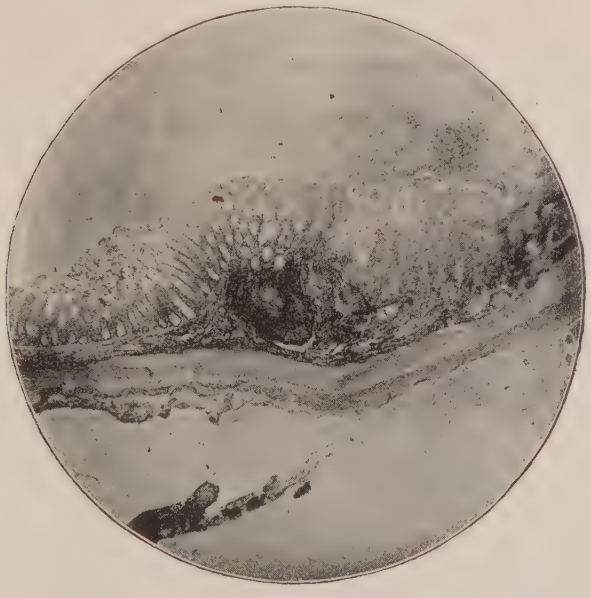


FIG. 59.



Acute follicular enteritis. Fig. 58, Swelling of intestinal wall due to infiltration of the submucosa with round cells (early stage). (× 32.) Fig. 59, Follicular ulcer produced by necrosis and extrusion of a follicle (late stage). (Photomicrographs by Professor Baginsky.)

FIG. 60.



Acute follicular enteritis. Inflammatory swelling of a follicle which is infiltrated and distended by round cells (early stage). (× 20.) (Photomicrograph by Professor Baginsky.)

time the stools become slimy, streaked with blood, contain little feculent matter but often scraps of curd and other undigested particles of food. The abdomen is distended, tympanitic, and a little tender in the course of the colon. In the more acute forms, to which the term acute dysentery is sometimes applied, the tenderness may be much greater and the pain severe. Later, the motions, which are passed at very frequent intervals, become small, brown or slightly bloodstained, and are often extremely offensive in odor. Tenesmus is, in many cases, a source of much distress.

Acute summer diarrhœa is the term commonly applied to the numerous cases of gastro-intestinal disorder attended by diarrhœa and vomiting which occur among children in large numbers, in temperate climates, during the warmer months. The attacks differ in character and vary greatly in severity. The common factor is the influence of a high air temperature sufficiently long continued to raise the temperature of the earth at a depth of 4 ft. to 56° F. This is attended by a great increase in the number and severity of the cases of acute gastro-enteritis such as may be encountered at any time of the year, and often by the occurrence of severe types which are hardly, if at all, to be distinguished by their clinical course from Asiatic cholera. Cases of every intermediate degree of severity may be met with during periods of epidemic prevalence of diarrhœa. Taking all forms together, acute summer diarrhœa is the cause of a mortality among infants and children which is always large, and is in some years and in some localities enormous. Nearly half the cases occur in children under five years.

Acute summer diarrhœa is favored by overcrowding and want of ventilation, and its incidence is most severe when the unhygienic conditions are combined with fouling of a porous soil, and the accumulation of dirt within and filth without the houses. A high summer temperature produces its effect: (1) by its depressing influence on the bodily powers, and the risk of chill while perspiring, or at sundown (a large number of cases begin during the night); (2) by stimulating bacterial growth in the soil and, possibly, in water; and (3) by increasing the rapidity with which various processes of decomposition and fermentation occur in food, especially milk. Frequently (1) and (3) appear to combine to produce the attack.

The relation of acute summer diarrhœa to microbial activity is, undoubtedly, intimate. Lesage has described a special group of cases characterized by green stools, which he attributes to infection of the intestines by a specific chromogenic organism. Flügge has isolated a spore-bearing bacillus which he believes to have a specific pathogenic action. It is anaërobic, breaks up proteids with the production of toxic bodies capable of causing diarrhœa directly, and is very resistant. Baginsky, who has given much study to this subject, has

come to the conclusion that there is no one specific microbe but that even in the most acute cases many saprophytic micro-organisms, having the power of producing decomposition, are present, and that in the intestinal canal they are pathogenic.[1] Accepting this view, the fact that the large majority of infants who suffer are hand-fed leads us to the conclusion that the microbes, as a rule, enter the alimentary canal with the food, which is usually cow's milk or some mixture containing it. Further, in warm weather especially, the milk may, before it is swallowed, already have reached a rather advanced stage of decomposition. Thus it may be found to contain lactic, formic, acetic, and butyric acids. Further, proteid decompositions may have occurred with the production of poisonous bodies, which have been shown experimentally to produce vomiting, purging, and in doses sufficiently large, collapse and death.[2] Lastly, there is reason to believe that the ingestion of decomposing food, or the catarrh which it excites, may cause certain microbes, which in health are harmless occupants of the intestine, to become pathogenic, especially the *b. coli communis.*

[We would emphasize the importance of the relation between bacterial activity and the intestinal affections of infants. While we cannot yet attribute to any specific organism a characteristic group of symptoms, it is along bacteriological lines that we must look for a rational classification of these intestinal disorders.

Marfan maintains that all the organisms held responsible for gastro-intestinal disorders of nurslings are found under normal conditions in the digestive tract, but that under certain abnormal conditions, they become pathogenic. As already stated, most commonly are found the bacillus coli communis, and the b. lactis aërogenes, and more rarely the streptococcus, b. pyocyaneus, staphylococcus and certain peptonizing bacteria, tyrothrix, b. mesentericus. Under pathological conditions it is rare to find pure cultures of any of the above organisms; "polymicrobism" is the rule. The most common combination is the colon-bacillus and the streptococcus.

The *bacillus coli communis* is undoubtedly but one member of a group of bacteria, the individual differences of which are so slight that we cannot at present differentiate between them. It is a constant inhabitant of the intestinal tract, being found most abundantly in the lower part of the small intestine and in the colon. Under normal conditions it is non-pathogenic and causes an acid fermentation. Under abnormal conditions, *e. g.*, improper feeding, it becomes pathogenic, its pathogeny being due to irritating substances in its protoplasm, as has been proved by Pfeiffer, Kolle, Löffler, and Abel.

[1] "Lehrb. d. Kinderkrank.," Berlin, 1896, 5te Auf., S. 775.

[2] Conf. Vaughan, *Trans. Amer. Ped. Soc.*, 1890, p. 109, and Baginsky, *loc. cit.*, S. 782.

The acid fermentation in the intestines then becomes excessive, causes an intestinal catarrh, and we have the three factors, which, according to Marfan, favor the invasion of the general system by this organism: (*a*) injury to the intestinal mucous membrane, (*b*) dimunition of the natural alkalinity of the body fluids, one of the natural defences against bacteria, and (*c*) increase in the virulence of the microbe.

Microscopic examination of the stools, careful study of cultures of the colon bacillus, of its virulence and its serum reaction, fail to explain its rôle in the gastro-enterides of nurslings, nor can any special symptom or special group of symptoms be attributed to its action. Its part in these affections is the more difficult to explain on account of its frequent association with other micro-organisms.

Its ubiquitous nature is well known, but whenever found, as is so common, in various parts of the body, it is almost invariably under conditions preceded or accompanied by intestinal trouble. Thus the organism is found in the bones of rachitic children, though rarely in pure culture, the streptococcus, the b. pyocyaneus and the staphylococcus usually being associated with it. Cultures from the bones of non-rachitic children are generally sterile. The frequent occurrence of intestinal troubles in rachitis is well-known. The organism also frequently invades and causes inflammation in the appendix, bile-ducts, kidneys, bladder, and sometimes the urethra. McFarland states that it has also been met with in puerperal fever, Winckel's disease of the new-born, endocarditis, meningitis, broncho-pneumonia, pleuritis, and chronic tonsillitis. While it cannot be conclusively proved that the colon bacillus plays the principal part in these infections, it undoubtedly does play an important part. It has been maintained that most of the summer diarrhœas of infancy and cholera infantum are due to the colon bacillus, but here again other micro-organisms frequently found in the stools undoubtedly play some part in causation, either directly through their own pathogenic action on the patient, or by enhancing that of the colon bacillus.

The *bacillus lactis aërogenes* Welch regards as one of the colon group. It is "characterized chiefly by its plumper form, its more energetic gas-production, its rapid coagulation of milk and its denser growth in cultures." (Mallory and Wright.) It is found most abundantly in the upper part of the small intestine.

The *streptococcus* is found in two varieties as described by Escherich: the *S. brevis* and the *S. gracilis*. A third, the *S. pyogenes vulgaris*, has also been described. The S. brevis has been found in the stools of nurslings, the gracilis in the meconium. In addition to these, Escherich has described a special organism which he calls the *enteritis streptococcus*. He has observed it in the sediment of cow's milk, and only in bottle-fed babies. It is found most abundantly at

the end of the ileum and at the beginning of the large intestine. Escherich believes three different clinical pictures may be attributed to this variety of the streptococcus: (1) benign, (2) toxic, (3) infectious, the last being the most serious and showing the microbe in cultures from the blood and urine. Its specific action however cannot yet be regarded as proved.

The enteritis streptococcus has been found in the lungs apparently causing broncho-pneumonia, in one case of purulent pleurisy, in the viscera where it is often associated with the b. coli, also in the kidney and spleen with the b. lactis aërogenes.

The *staphylococcus pyogenes albus* is often found in the first few drops of mother's milk, gaining access to the nipple from the neighboring skin, and under normal conditions is non-virulent. In mastitis, however, it becomes virulent and causes gastro-enteritis in the nursling.

The *bacillus pyocyaneus* is a rare inhabitant of the intestines under normal conditions. It occurs under pathological conditions, however, and the clinical symptoms associated with it are severe. It is not found in milk, and probably gains access to the intestinal tract from the hands of hospital attendants, and also from the pus of an otitis or rhinitis, in which it often occurs. Welch states that it is often found in diarrhœa and dysenteric discharges, and that it may cause hæmorrhagic and necrotic enteritis.]

The majority of cases of acute summer diarrhœa are examples of acute gastro-enteritis, but in those most acute cases designated by the term **cholera infantum** death may ensue at an early stage of the morbid process. In such cases the intestines contain only a little opalescent or creamy mucus. The mucous membrane is hyperæmic in patches, and even in cases of only a few hours' duration, there is denudation of the epithelium both in the stomach and intestines.[1] The solitary and agminated glands and the mesenteric glands are enlarged. There is fatty degeneration of the liver cells, and nephritis, parenchymatous and glomerular, which may be intense even in cases of very short duration. The lungs show areas of collapse with commencing pneumonia. The blood may be inspissated and coagulate imperfectly, but a consideration of the morbid anatomy would alone be sufficient to prove that the symptoms are due to general toxæmia, and cannot be attributed, as was formerly the custom, to the mere draining away of fluid through the intestines. In the most acute cases, indeed, and those most rapidly fatal, there may be very little diarrhœa.

In a well-marked case the symptoms run a rapid course. The infant or young child, who has previously been in good health or has

[1] Klein, *Diarrhœa and Diphtheria, Supplement to Report of Medical Officer, L. G. B.*, London, 1889 (C.—5638), pp. 14, 78.

suffered for a few days from malaise and slight dyspeptic symptoms, is seized with severe vomiting and diarrhœa. Any food which it may be induced to swallow is rejected almost immediately. The stools, which may or may not be numerous, are at first yellow, and contain the remains of food. Soon they become quite fluid, and of a brown color. The color grows quickly less deep and the motions more transparent, until finally they consist of a colorless, slightly opalescent liquid, which does not stain the napkins. The temperature is generally raised, and is often very high—105° F., and even 107° F. The child is restless, changing its attitude constantly, and kicking. When at rest it lies with its legs drawn up. It looks extremely ill; the face is flushed; the skin of the abdomen, which may be slightly distended, can be pinched up into folds—"like linen," as has been said. After a short time, usually a few hours, collapse sets in; the temperature falls to the normal, or below (97° or 96° F.); the extremities are cold; the face is pale and drawn. The eyes—sunken, half open, and motionless—are surrounded by dark rings; the lips and the ears are cyanotic. The abdomen is retracted; the skin retracted over it. The fontanelle is collapsed, the tongue dry, the breath cold, and the respiration slow or irregular and labored. The pulse is small and rapid, often imperceptible at the wrist; the heart's action feeble. Diarrhœa and vomiting cease, or are very rare, and the child lies in a condition of the most extreme apathy—generally on its back with the legs extended, motionless but for the irregular, shallow respiration. Death may, and when collapse is well developed does usually, ensue at this stage. On the other hand, the symptoms of collapse may be much less severe, and give way more or less rapidly to those of reaction, with secondary fever, in which the temperature presents a morning fall and evening rise. The child remains in a condition of hebetude, is with difficulty roused, or, if beyond infancy, suffers from low muttering delirium. The face has a dusky flush; the eyes are suffused; the tongue dry, coated, and tremulous. Diarrhœa is not usually a prominent symptom, but everything taken by the mouth tends to excite vomiting. In other cases, and these form, perhaps, the majority, reaction is never properly established, and the child remains in a condition of collapse for several days. A sudden rise of temperature usually precedes a fatal termination.

If the child recover it is liable to suffer further attacks, acute or subacute, which are often determined by a chill.

As *complications* of gastro-enteritis, bronchitis, broncho-pneumonia (generally basal), and secondary nephritis may be enumerated. In infants convulsions are not infrequent, and, according to Jacobi, are in many cases due to nephritis. Irritated by the frequent stools, the buttocks and perineum become inflamed. Intertrigo, impetigo, and

pustular eczema are produced, and, owing to want of care, extend often to the thighs, back, and abdomen, and even to the upper limbs, face, and scalp.

[**Prophylaxis.**—We may do much to prevent the development of intestinal trouble or to prevent its further progress once developed. The child's general condition should be kept as good as possible by attention to his diet and general hygiene. Detailed reference to the management of the diet, in both breast-fed and artificially-fed infants has already been made and is pertinent here. (Appendix to Chapter IV.) Great care should also be paid, especially in summer, to the diet during the second and third years of life. If living in the city, the milk supply should be rigidly overlooked. Much good has been done in this direction in Boston, New York, and Chicago by the establishment of "milk funds," by means of which a supply of pure milk is insured to the children of the poor at a minimum expense.

Almost equally important with the diet are the general hygiene and personal cleanliness of the child. Abundance of pure fresh air is essential to the maintenance of good health, and where removal to the country is impossible, parents should be urged to take their children as much as possible to the parks. They should also be directed to give the daily bath and to exercise great care in the immediate removal and washing of soiled diapers.

The practice of allowing a diarrhœa during teething cannot be too strongly condemned and should be relegated to the domain of ignorant nurses and midwives. It is but preparing the soil for invasion of some serious form of intestinal trouble.]

In the **treatment** of a case of acute gastro-enteritis in an infant fed by hand, milk should be stopped, and the patient should be allowed to drink freely of water (boiled) cold or hot, to which some Vichy water may be added. As food, whey, weak veal broth, or egg-water may be given in small quantities at frequent intervals; or, except in the youngest infants, barley, wheat, or oatmeal water, which, when properly made, has the advantage of containing very little fermentable material.

[*Egg Water.*—The white of an egg stirred into 4 to 6 fl. oz. of boiled water, and sweetened with white sugar or a solution of milk-sugar.

Whey.—After the milk has been curdled with rennet, the curd should be beaten up with a fork and the whey strained off through muslin. *White Wine Whey* is made by adding 2 fl. oz. of sherry to half a pint of milk just at the boiling point. The mixture is then boiled for two minutes, and afterwards allowed to cool in a basin. The whey may be poured off, or strained off as directed above. Appendix.]

When vomiting is an early and prominent symptom, the attack

may sometimes be cut short by washing out the stomach with boiled water, at 98° F., to which resorcin (1 in 1,000) or boric acid (½ per cent.) may be added. Before withdrawing the tube, castor oil (℥j) may be introduced into the stomach. If vomiting recurs, the washing may be repeated, and, in an infant of nine months, tincture of opium ♏j or solution of cocaine (5 per cent.) ♏ij–iij left in the stomach. If the stomach be not washed out, the treatment should be commenced by a dose of castor oil or, perhaps better, of calomel (gr. ⅛–¼) every two or three hours to four doses. If diarrhœa be present from an early stage, castor oil or calomel should equally be given to clear the intestines, and the attack may subside. If, however, the stools continue to be watery and foul-smelling, an antiseptic, such as naphthalin or salol, will be preferable; the latter, which is resolved into phenol and salicylic acid in the upper part of the small intestine, is perhaps the more useful in the early stages. Either drug should be given in small doses frequently repeated. If the stools are green, "like chopped spinach," and alkaline or faintly acid, lactic acid (♏j–ij in dill water) is indicated. Watery, grayish-brown stools may be checked in some cases by calcium phosphate (gr. v–viij). When the temperature remains high, with flushed face and distended abdomen, or if symptoms of collapse threaten, especially if small mucous stools are passed, large clysters are indicated. In an infant nine to twelve months old about a pint should be injected slowly, preferably by means of an irrigator. As a rule, the clyster is retained for from half an hour to two or three hours, and is then evacuated along with the infective contents of the large intestine. In addition to thus removing poisonous matter, these injections may have considerable effect on the temperature. Thus a pint at 85° F. may produce a very rapid fall from 103° or 104° F. to below normal, and at 92° F. may cause a fall of several degrees. Such injections must therefore be given with caution, and their effect watched. Unless it is desired to reduce the body temperature rapidly, the temperature of the enema should be 97° or 98° F. as it enters the rectum; and to attain this, that of the fluid in the reservoir should be 1° or 2° F. higher. The fluid may be medicated in various ways—with boric acid (0.5 per cent.), sodium chloride, or tannin (0.5 per cent.), or with lime-water (equal parts). As an alternative treatment, Heubner recommends small enemas of salicylic acid (gr. j to ℥j) or quinine hydrobromate (1 per cent.). In acute cases, or if there be severe colic, it is well to apply hot fomentations or a light poultice to the belly, which should afterwards be covered with cotton-wool or a flannel binder.

[The treatment should be prompt and rigidly enforced, no matter how slight the attack. Two factors must be borne in mind: the original source of intestinal trouble, *i. e.*, the food, and the condition

of the digestive tract. The infectious agent has been introduced by the food, which, both in the body and out of it, furnishes an excellent culture medium for the development of bacteria. The intestinal tract contains much toxic material the result of proteid decomposition, even in the earliest stages of the disorder, before inflammatory changes have been established. Obviously our measures must be directed to the correction of these two conditions, in other words, stop the food, clean out the intestinal tract. All food should be withheld for at least twenty-four hours. If the patient be breast-fed the breast must be relieved by use of the breast-pump. Sterilized water should be given freely and often. The intestinal tract should be attacked by mouth and rectum, laxatives being given and copious irrigation of the colon being performed. Calomel is by far the best intestinal laxative we have, and has the additional advantage of some antiseptic action. We prefer it, however, in doses larger than those advised by the author, one grain every four hours being given to a child eight months old until he has received three or four doses. There is little if any danger of salivation, as these doses take care of themselves by the free evacuations which they cause. The lower bowels should be flushed by high enemata of plain warm water, one, two, or three times daily. The technique is important and should be carried out either by the physician himself or by a skillful nurse. Place the child on his back, introduce a large rubber catheter, or small rectal tube attached to the tube of a fountain syringe, an inch or two within the anus and start the water flowing; gradually push the catheter up into the bowels, in this way the water distends the bowels and the catheter is easily pushed on. As much as a gallon should be used, the water flowing back and out alongside the catheter.

There are few infants and children who cannot stand the starvation-and-evacuation process outlined above for the first twenty-four hours, provided this treatment can be instituted early, within the first day or two. The importance of early treatment in these cases cannot be exaggerated. Under this simple régime, the majority of cases of summer diarrhœa in otherwise healthy and well-nourished children will subside in a day and the patient be as well as ever in two or three days. Unfortunately, however, none but the most intelligent mothers can be persuaded to "starve" their babies for that first twenty-four hours, and often we have to allow a-to-them "food." To such we must then give a food which will cause the least possible harm in the intestinal tract. Mention of the evil effects of proteid decomposition has already been made. Obviously milk and all proteid-containing foods should be withheld. Starchy foods only may be given and we may use rice water or arrow-root 4 to 8 ounces three times daily. Sterilized water should be given freely between feedings.

The result of the above measures carried out for twenty-four hours

is, as a rule, increase in the number and putridity of the stools due to the action of the calomel in producing free evacuation of much of the decomposed intestinal contents, and improvement in the nervous condition of the patient due to the checking of absorption of toxic material from the intestinal canal.

The subsequent treatment consists in a gradual resumption of the usual diet and continued disinfection of the intestinal canal. We must remember that one of the results of the attack is to impair digestive power, and that hence too much work should not be thrown suddenly onto the digestive functions. The number of breast-feedings, started with two or three in the twenty-four hours, may be gradually increased, the starchy food being correspondingly diminished. If bottle-fed, modified milk with low percentages should be prescribed. Especially shall we find it imperative to begin with a minimum amount of proteids, often as low as $\frac{1}{4}\%$ of this troublesome element. The percentages should be increased as rapidly as possible, that we may give the child a diet sufficiently nutritious. For medication, we may give after the first twenty-four hours, calomel gr. $\frac{1}{10}$ every four hours, or naphthalin or salol as mentioned above. Daily irrigation or at least rectal enemata should be continued. The time and rapidity of resumption of the usual diet must of course vary with the case and no hard and fast rules can be laid down in this respect. We must be governed by the general condition of the patient, the degree of his nutrition and the character of the stools, especially their odor and color. The increased putridity of the stools during the first twenty-four hours usually diminishes and disappears in the next twenty-four hours. If the patient be in a robust condition it is wiser to withhold his proteids until the stools have lost their putrid character, as his nutrition can stand the limited diet for that length of time. On the other hand we must not subject a weak and emaciated infant to a too rigorous diet, but must run the risk of increased toxæmia from decomposing proteids in order to preserve nutrition. Such infants of course have less digestive power than the robust cases from whom we withhold proteids, but we must nevertheless take our chances, carefully regulating and modifying the diet, adapting the food to the limited digestive powers. Predigestion by peptonization is often helpful. It is in such cases that feeding by accurate percentages is invaluable.]

The treatment of *cholera infantum* and the most acute cases of summer diarrhœa must, in their earliest stage, be the same as that already indicated. When the characteristic stage of collapse is established, drugs given by the mouth have little effect, since absorption is very slow, but small doses (gr. $\frac{1}{10}$) of calomel may be given, or naphthalin or salol may still be of use owing to their local antiseptic action. The use of large warm antiseptic clysters should be persevered in; they are perhaps especially indicated when the urine

is suppressed or passed in very small quantity. Hot drinks may be given if the child will swallow, and, if they induce vomiting, need not be discontinued unless they appear to add to the exhaustion. Three or four hot baths should be given in the course of twenty-four hours, or hot packs with or without the addition of brandy to the water used. In the intervals every effort should be made to keep the child warm by artificial means. As the gastro-intestinal secretions are arrested more or less completely, only such foods as whey and broth can be expected to be of any service. If reaction occur and remittent pyrexia be established, such drugs as quinine, salol, naphthalin, or if there be much diarrhœa, bismuth may be given with more expectation of a favorable result. High temperature may call for the use of cool or cold packs.

[If we can begin treatment early enough in cases of gastro-enteritis, seldom shall we have a true cholera infantum. This term, used loosely and indefinitely, should be applied only to those cases with a true choleriform diarrhœa. The onset of such intestinal infection is rarely sudden. It is almost always preceded by more or less digestive disturbance, generally diarrhœa of a few days' or weeks' duration. Such digestive disturbance but weakens the resisting power of the intestinal tract and thus prepares the way for an invasion of some more serious infectious agent, and cholera infantum develops.

Here we are dealing really with a case of general systemic poisoning, and we must strike at the root or source of the toxæmia and at the same time counteract the effects of the poison upon the heart and the nervous system. The first object we strive to attain by immediate evacuation of the stomach and intestinal tract by stomach washing and high irrigation; cathartics are of but little value as they are apt to aggravate the vomiting, their absorption is uncertain and thus valuable time is lost. For the second object, morphine is undoubtedly our best weapon, and the method recommended by Holt most effective. He advises giving hypodermically, morphine gr. $\frac{1}{100}$ and atropine gr. $\frac{1}{800}$, to a child one year old, to be repeated every hour till arrest of the vomiting and purging and improvement in the heart's action are attained.

On account of the great loss of fluid by the discharges, water should be given freely; by the mouth if possible, or by hypodermic or intra-venous injection. The high temperature should be combated by baths and ice-cap to the head, the baths being given every hour or two, beginning at a temperature of 100° and gradually lowering this by the addition of ice to about 80°–85°. Stimulants must be used freely, and often hypodermically; brandy is our best stimulant. Food must be withheld until the vomiting has ceased or at least lessened.

The prognosis in the most severe cases of cholera infantum is bad, the majority terminating fatally, regardless of the treatment pursued. These results emphasize the necessity of prophylaxis in, and prompt energetic treatment of, even the slightest case of summer diarrhœa in infancy.]

CHAPTER XXXII.

CHRONIC DISORDERS OF THE GASTRO-INTESTINAL SYSTEM.

Chronic Gastro-enteritis—Dilatation of the Stomach—Infantile Atrophy—The Hydrocephaloid Condition—Congenital Stenosis of the Pylorus—Constipation—Prolapsus Ani.

Chronic gastro-enteritis is frequently a sequel of the acute form. The child becomes much emaciated, the abdomen is large, baggy, and soft, though liable to be rendered tense by flatulence. The appetite is capricious, sometimes ravenous ; at other times there is complete anorexia. Thirst is generally a distressing symptom, and is due in part, at least, to slight general catarrhal stomatitis, which is present in many cases. The tongue is small, raw, or presents the peculiar form of superficial catarrh to which the term geographical tongue or epithelial desquamation is applied.

Dilatation of the stomach is a common complication, especially in rickety children ; it is not rare in infancy, when it is very apt to be associated with urticaria, sometimes with convulsions. The dilatation is caused by the distension of the stomach produced by the habitual use of starchy foods determining flatulent dyspepsia, and is favored by bulky meals. The chief symptoms are vomiting, commencing shortly after food and several times repeated, thirst, and epigastric uneasiness unrelieved by food. The abdomen is distended and tense, and the percussion note is very tympanitic over the left hypochondrium and down to, or below, the umbilicus. Similar physical signs are produced by dilatation of the colon, and the two conditions may be combined. When a certain diagnosis is necessary, which is rarely the case, it may be made by giving the child first one part and then the other of a seidlitz powder—the rapid distension of the stomach produced by the liberation of carbonic acid gas causes a marked temporary alteration of the area of tympanites ; by auscultatory percussion the note will be observed to change very markedly when the finger passes beyond the stomach.

When chronic gastro-enteritis is well established, and the colon has become more or less involved, the bowels act frequently, and the motions consist mainly of brown mucus, often streaked with blood, or of gelatinous material like white of eggs, with perhaps scraps of

undigested food. The stools have not the ordinary fæcal odor, but are very offensive, sometimes horribly so, recalling the odor of putrid meat. In very many cases the stool is passed shortly after the ingestion of food, the entrance of which into the stomach determines a rapid and often painful peristalsis. To this combination, so frequently met with, in which foul mucoid stools are passed after each meal, the term lienteric diarrhœa is commonly applied. The child is restless and fretful, but has occasional intervals of drowsiness, which are to be attributed in part to the loss of sleep at night, and in part to the absorption of toxic matters from the intestine. In some cases the large intestine is the part most affected. After an attack of gastro-enteritis, which may not have been very severe, the child begins to suffer from tenesmus, and passes foul mucous motions, some of which contain small scybala. The tenesmus may be almost continuous, and small quantities of mucus are passed several times an hour. This condition is due to catarrhal inflammation of the *sigmoid flexure and rectum*. When the *colon* itself is the part involved the stools are less frequent, and there may even be constipation. Then the most prominent symptom is colic. The attacks of colic may or may not end in an evacuation, and during their continuance the colon may, in an emaciated child, be seen distinctly outlined through the abdominal walls. These attacks of colic are often determined by the ingestion of food, and the child is quickly reduced to a condition of great exhaustion and emaciation.

The *morbid changes* in the stomach and intestines vary with the severity and duration of the disorder. In the milder forms, though the stomach is almost always dilated, its mucous membrane may show little change to the naked eye, beyond perhaps some areas of congestion or pigmentation of the mucous membrane, which is covered by a layer of tenacious mucus. In a more advanced stage the mucous membrane loses its elasticity, and becomes opaque, though in the most extreme cases the atrophy may be so great that the stomach walls are so thin as to be transparent. The earliest lesion is a glandular degeneration of the epithelial cells, and an inflammatory infiltration of round cells into the subjacent connective tissue. This round-celled infiltration extends later on into the muscularis mucosæ, while at the same time the infiltration at first produced undergoes organization into fibrous tissue. This, in its contraction, throws the surface of the mucous membrane into irregular folds, and distorts or occludes the glandular tubules. At the same time the secreting epithelium undergoes degeneration. In the final stage there is extensive cirrhosis of the mucous membrane, and complete destruction of its glandular structures. Usually these changes do not affect the whole extent of the mucous membrane in a uniform manner, but are most advanced in the neighborhood of the pylorus.

The small intestines undergo a similar process of interstitial inflammation with consequent fibrosis. The gut is usually found collapsed and nearly empty, and it is common to find in the ileum, especially near the ileo-cæcal valve, extensive shallow ulcers, which are usually longitudinal. In the latest stage the intestinal walls are so thin as to be semi-transparent. The large intestine is not infrequently distended with gas; its mucous membrane shows patches of punctiform pigmentation due to minute hæmorrhages, and in a large percentage of cases follicular ulceration is present.[1] Interstitial inflammation here also leads eventually to fibrosis and thickening of all the coats of the colon. The gut may thus become greatly dilated and thickened. In some cases, in which this *dilatation of the colon* attains enormous proportions, the condition has originated very early in life and may perhaps have been due to congenital defect. Corresponding to the progressive deterioration of the glandular structures there is a progressive diminution of the digestive powers, and progressive increase in the anæmia and emaciation. Gastric digestion is much prolonged, so that the stomach, instead of being empty in two hours or less, may still contain remnants of a meal (milk) taken five hours before.[2]

The tendency of the disease, except in the most extreme stage, is towards recovery; but the liability to recurrent attacks or exacerbations is great, and in making a *prognosis* regard must be had to the care with which instructions as to the clothing and feeding of the patient are likely to be carried out. A large number of children who have partially recovered are carried off by whooping-cough or by measles, which in these patients is very apt to be complicated by severe mucous diarrhœa due to an exacerbation of the intestinal catarrh. In an infant which has become much emaciated and anæmic from gastro-enteritis, the prognosis is extremely bad. The *diagnosis* is not commonly difficult, though it may be impossible always to exclude the possibility that the condition may be due to tuberculosis. Cases of this kind are very often spoken of as "consumption of the bowels"; and the use of this term being assumed to exclude all hope of recovery is sometimes made an excuse for neglect. As a matter of fact, however, tuberculous enteritis is very uncommon in infancy and childhood, and seldom occurs without the presence of symptoms or physical signs indicating involvement of other organs.

In the **treatment** of chronic gastro-enteritis, attention must be directed to the feeding and clothing of the child, and means must be taken to promote the elimination of the putrescent material in the intestines and to prevent decomposition by improving the digestive powers and administering antiseptic and carminative drugs. When

[1] In 62 per cent., according to W. Soltau Fenwick, to whose Report (*Brit. Med. Journ.*, 1896, vol. ii., p. 829) I am much indebted.

[2] W. S. Fenwick, *loc. cit.*

the case is first seen, milk should be replaced by a diet consisting of egg water, whey, or meat juices, to which carbohydrate foods, such as fine wheatmeal or oatmeal, may be added with caution. When the child begins to improve a milk diet may be resumed, the effect being carefully watched. In children who have previously had a mixed diet, it may sometimes be at once replaced by the exclusive use of milk diluted with water, or barley water. The child should wear woollen garments next the skin, and special care should be taken that the abdomen and thighs are covered, for these parts are often very insufficiently clad, not only in children of two or three years but even in those of eight or ten. The child should be placed under the best procurable hygienic conditions, should live in well-ventilated rooms, and should spend many hours a day in the open air. Treatment by drugs should be commenced by a dose of castor oil (℥j) or calomel (gr. $\frac{1}{2}$ at one year), followed by small doses of castor oil (♏v) or calomel (gr. $\frac{1}{12}$ to $\frac{1}{10}$) twice a day. After each meal a dose of pepsin and hydrochloric acid, or of papaine, should be given. In infants the papaine may be added to the food, and if eructations are troublesome at any age, it is best to give papaine with the food, and a few grains of sodium carbonate in dill water shortly after the meals. A method which, in careful hands, is in many respects superior is the use of predigested milk. Convenient powders for this purpose are sold, and the quantity of proteids can be varied by diluting the milk before digestion; while the quantity of fat can be increased, if desired, by the addition of cream.

[The accurate modification of cow's milk, either at the laboratory or by home methods (see appendix Chapter IV.) is attended with excellent results in the feeding of these cases. The condition is one of impaired general digestive power and by prescribing accurate percentages of fat, sugar, and proteids, we adapt the amount of these ingredients to the diminished fat-, sugar-, and proteid- digesting powers. It would seem more rational to exercise what digestive powers the child has, diminished though they be, by making them do the limited amount of work of which they are capable and thus favor their development, than to do this work for them by attempts at predigestion. It is the same principle upon which we proceed in seeking to develop a group of impaired muscles; we give these muscles exercise at first gentle and slight, gradually increasing it as the power of the muscles increases.

It has been the experience of the reviser that these cases of chronic gastro-enteritis do better by careful and painstaking attention to the diet along the lines indicated above than by reliance upon "predigestives," "intestinal antiseptics" and "anti-dyspeptics."]

An active extract of malt is a palatable remedy which acts well in many cases during convalescence. To correct the fœtor of the stools

antiseptics should be given, such as naphthalin, salol, resorcin (ʒj of a 1 in 1,000 solution). So long as the stools contain much mucus, it is, however, advisable to continue small doses of castor oil, which may be combined with resorcin, or the glycerine of carbolic acid (♏j–ij). If the stools be frequent, small doses of tincture of opium (♏ss–j) may in this later stage be added. Lienteric diarrhœa, after the preliminary administration of laxatives, should be treated by a mixture containing arsenic, strychnine, and the citrate of iron and ammonia, to which, if the diarrhœa persist, it may be necessary to add minute doses of opium. If the stools are copious, bismuth sometimes answers well, but its action is uncertain. Astringents are not to be recommended. As soon as the stools begin to lose their mucous character, some preparation of iron should be given, and none is superior to the citrate of iron and ammonia, which is well borne by children. It should be combined with tincture of belladonna (♏iv–v at one year) if there be abdominal pain or colic. At a somewhat later stage, the syrup of the phosphate of iron is usually well taken, but the patient should eventually be put upon a mixture containing cod-liver oil and iron, guarded, if there be still a tendency to diarrhœa, by opium. When convalescence has been established, the patient should be given the benefit of sea or mountain air.

When there is reason to suspect dilatation of the stomach attention should be given to the bulk of the meals; they should be small, and given at frequent intervals. Starch, and its derivatives, should be excluded. As a rule, improvement ensues after a few days, during which the child complains of being hungry. In cases of longer standing, especially those in which the vomiting takes place many hours after the meal, it may be necessary to have resort to washing out the stomach.

Infantile atrophy, marasmus, athrepsia are terms somewhat loosely applied to a condition of malnutrition in infants, which may be due to many different causes. In some cases the cause is to be found in congenital syphilis, or in tuberculosis. Others must be attributed to a diet insufficient either in quantity or quality; while in others, and these form the large majority, the marasmus is due to chronic gastro-intestinal dyspepsia or catarrh. Cases will, however, occasionally be met with which cannot with any confidence be assigned to any one of these categories, and it seems necessary to admit that certain infants are born with defective powers of digestion and assimilation. After death in such cases the intestinal walls are very thin, but there is commonly no visible hyperæmia, glandular swelling, hæmorrhage, or ulceration. The epithelial cells are smaller than in health, owing to a diminished quantity of protoplasm, and the number of leucocytes in the intestinal wall is small, very few being seen between the epithelial cells, where in health they are nu-

merous. There are fewer also in the adenoid tissue.[1] The condition, however, it must be admitted, resembles closely that produced by starvation, and the diagnosis must be made by a process of exclusion. If this be done with due care and discrimination, it will be found that the number of cases of so-called simple atrophy is extremely small. It is to be remembered that though it is rare to find a mother guilty of withholding deliberately the necessary quantity of food from her infant, examples of almost incredible ignorance are not infrequent. On the other hand, in the case of illegitimate infants "adopted" for a small consideration, criminal intention may exist, which will, of course, be concealed from the medical attendant.

The term **hydrocephaloid condition** is sometimes applied to the state of depression and collapse into which young children who have suffered long from severe chronic gastro-enteritis, or who have experienced a severe attack of acute summer diarrhœa or cholera infantum, frequently sink. The patient lies in bed, usually on the back, in a somnolent state. The eyes are half-open, and exude a little muco-purulent secretion; the pupils are large and sluggish. The face is sunken and the whole head, as it were, shrunken, so that the anterior fontanelle is collapsed, and the cranial bones are in apposition or override each other. The belly is soft, the skin inelastic and easily pinched into folds which disappear only slowly; the tongue is dry; and the lips covered with sordes. The respiration is shallow. It may be rapid but is more often irregular, sometimes presenting a distinct Cheyne-Stokes type. The pulse is irregular, small, generally imperceptible at the wrist. The temperature is subnormal, 95° to 97° F. The urine is scanty, and the child is indifferent to food and drink. The condition may deepen into coma, or the end may be brought about by convulsions. More often, perhaps, careful nursing in good hygienic surroundings leads to recovery, the respiration becomes more regular, the pulse improves, urine is again excreted freely, the bowels begin to act, usually with abnormal frequency, and the child again desires food, and observes events happening about it. The patient should be nursed in a warm, well-ventilated room. A warm bath should be given at once, and the child should then be enveloped completely in a soft blanket, leaving only the face free, and either nursed by the fire or placed in a cot with hot-water bottles beside it, the whole covered lightly with a blanket. A diffusible stimulant (*e. g.*, ammonia and ether), or a few drops of good whisky or brandy (℥xx in water), or champagne should be given every hour. Minute doses of strychnine also seem to be of use. No milk should be given; but, in its place, meat jelly or veal broth in small quantities every hour. The warm bath may be repeated four or five times a day.

[1] Heubner, "Penzoldt and Stintzing's Handbuch," Bd. iv., S. 107.

Congenital stenosis of the pylorus may be due either to a simple narrowing of the pyloric orifice or to hypertrophy of either the circular or longitudinal muscle fibres. The degree of stenosis varies. In the more pronounced cases there is a thickening due to hypertrophy of the longitudinal muscle fibres, distinctly limited above and below the orifice. The stomach becomes dilated, and secondary gastritis leads to some general thickening of the stomach walls and to infiltration of the mucosa and submucosa by small cells. The swelling of the mucous membrane produced by this secondary gastritis still further narrows the pyloric orifice.

The severity of the **symptoms** is proportionate to the degree of narrowing. The most characteristic are obstinate vomiting after ingestion of food, even when the meals are very small, and the passage of small constipated stools. The symptoms may not appear until a short time after birth, and the vomiting at first may occur only after large meals. Subsequently the symptoms resemble very closely those of chronic gastric catarrh, with which, as has been said, stenosis is usually complicated at an early stage. The dilatation of the stomach which ensues diminishes its digestive power, and favors fermentative dyspepsia. In some cases the thickening of the pylorus has been felt as a small cylindrical tumor above the umbilicus. The interference with digestion involves a failure of nutrition, emaciation, and progressive loss of strength. Death may ensue within a month in extreme cases. In the less severe life may be prolonged for two years at least, but for how much longer is an interesting question not yet determined.

Treatment must be directed mainly to prevent or alleviate catarrh. The meals should be small, and if the infant be suckled, as is preferable, special directions must be given to this effect. Pepsin or papaine will be of use to supplement the imperfect digestive powers. In hand-fed infants the milk should be predigested, and starchy foods should be excluded; meat juice may be given by the mouth, or preferably by nutrient suppository. In any case in which the existence of a pyloric tumor can be detected, the propriety of an operation with the view of dilating or excising the pylorus would arise for consideration.

Constipation.

An infant at the breast has during the first two months of life from two to four, generally three, motions in the twenty-four hours; from two to seven or eight months old, two or three motions; at one year old, one or two motions. The motions are of a bright orange color, soft but not liquid, and stain the napkin. So long as the motions retain these physical characters, diminished frequency is not

a source of inconvenience, and an infant who passes only one such healthy motion a day should not be considered constipated.

Constipation—that is, the passage at unduly long intervals of firm, pasty, or hard stools, generally altered also in color—is extremely common during the first two or three years of life, and is the source of much distress. It is to be traced, as a rule, either to error in diet or to a peculiar conformation of the lower bowel, but certain other conditions call for a brief notice.

Retention of meconium may be due to inspissation to congenital stricture, or to occlusion of the intestine. The former condition is easily relieved by a simple enema; but it should be remembered that no meconium may be passed after birth, owing to the intestine having been emptied either into the amniotic fluid or the maternal passages during difficult labor. Congenital occlusion occurs in the rectum, or

Fig. 61.

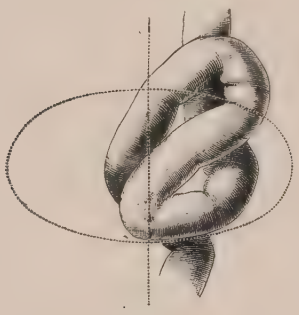
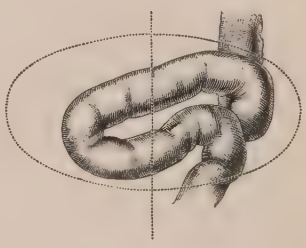


Fig. 62.



Semi-diagrammatic drawings to illustrate the three chief types of abnormality of the sigmoid flexure, which are the source of habitual constipation in infants. Fig. 61, Ascending position; Fig. 62, transverse position.

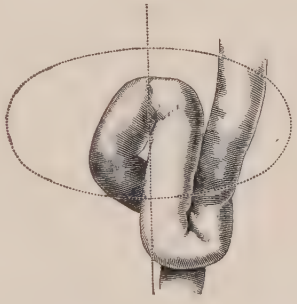
at the anus, where it may be diagnosed by physical examination, or in the upper part of the intestine, jejunum, duodenum, or ileum, when diagnosis is difficult, if not impossible. The first-named condition is due to a developmental defect; the second, probably, to fœtal peritonitis or enteritis.

Symptomatic constipation accompanies (*a*) fever when it is attributed to arrest of secretions, and is apt to be followed by diarrhœa due to enteritis set up by putrid decomposition of the retained fæces; (*b*) nervous diseases, such as meningitis, when it is believed to be due to defective peristalsis; or (*c*) peritonitis.

Alimentary constipation is observed in infants fed on cow's milk. It is characterized by the passage once a day, or once in two or three days, of large, pasty, firm, or hard whitish motions, which consist largely of undigested curd. It has been attributed to the excess of

casein, to the excess of earthy salts, to the poorness in sugar, and to the poorness in fats, characteristic of cow's as compared with human milk. To these conditions may be added, if the milk be insufficiently diluted, a diminution in the quantity of water, at any rate in proportion to the total solids. It is probable that all these causes contribute, but that the last two are the most important. At any rate, addition of fat to the diet will often relieve the condition. Premature use of a starchy diet is also a cause of constipation in infancy, but this error, and in older children other errors of diet, whether the too exclusive use of cow's milk or of starchy foods, leads usually to a condition in which constipation alternates with diarrhœa, and is soon associated with flatulence, dilatation of the stomach and intestines, and habitual distension of the abdomen. The retention of hardened fæces sets up catarrh of the mucous membrane of the intestine. This appears at first to favor constipation by coating the fæcal masses with slippery mucus, but later, when more intense, it determines diarrhœa. The exposure of the surface of the abdomen to cold favors if it does not determine the production of this condition of constipation, with intercurrent attacks of diarrhœa due to catarrh. Many infants after being taken out of long clothes, and indeed the majority of children down to the age of three or four years, are insufficiently clad about the abdomen.

FIG. 63.



Descending position (from MARFAN after BOURCART).

Anatomical (**essential**) **constipation** is due to an excessive length and coiling of the sigmoid portion of the colon (see Figs. 61, 62, 63). The symptoms vary with the degree of the congenital defect, and are aggravated by those errors of diet which tend to produce constipation. The history is generally clear; the patient has always had motions at infrequent intervals, and they have generally been passed with difficulty and much straining; they have been of the consistence of a firm paste which does not stain the napkins, or formed "like a grown-up person's" or have been small, hard, rounded masses, often coated with mucus and, perhaps, streaked with blood. The child has little or no pain except during defæcation, and laxatives either produce no effect or a single motion, which is accompanied by great pain and distress. The intervals between the motions may be very long; such periods as three or four days are common, and a week or

ten days not uncommon. As a rule, the child does not present other definite symptoms of illness, but it is commonly anæmic and ill-nourished, seldom fat. A tumor may be felt deep in the left iliac fossa, and a fæcal mass may be felt by the finger in the rectum. The anus may be excoriated or fissured, thus accounting for the streaks of blood on the fæces, and in part for the pain on defæcation, which is often so great that the child voluntarily represses the call to pass a motion, and thus aggravates the condition from which it suffers. The violent straining during defæcation may produce prolapsus ani, or hernia, especially of the umbilicus.

The remoter consequences of habitual constipation when obstinate are undue excitability of the nervous system, produced probably by absorption of products of decomposition from the alimentary canal, and showing itself in irritability, restlessness, broken sleep, and occasionally by convulsions. Frequently there is urticaria, which is liable to become complicated by impetigo, and to lead, on the buttocks and about the anus and groins, to the condition commonly called eczema of these parts. Retention of fæces in the colon leads to catarrh of its mucous membrane, which will be aggravated if the belly is not warmly clad. The catarrh may be succeeded by ulcerative colitis. When extreme and of long standing, congenital constipation may lead to great dilatation and hypertrophy of the colon,[1] or to intestinal obstruction.

Treatment.—The motions of a healthy infant are generally passed soon after suckling, and when there is a tendency to constipation advantage should be taken of this physiological fact. It is hardly necessary to insist that habit and custom have a great deal to do with the regularity of the act of defæcation, and a skilful nurse will begin the establishment of this habit as early as the fourth or fifth month of life by soliciting the infant to pass a motion at regular hours by holding it in an appropriate attitude.

When called upon to treat constipation in an infant attention should first be directed to the diet and clothing. The belly and limbs should be covered completely. If the infant is fed by hand starches should be eliminated, and replaced by malted foods made with dilute milk, to which milk-sugar or, in default, cane-sugar is added. If cream of good quality can be obtained it may be added to the milk, or given separately in a teaspoon, so that two to three or four drachms are taken daily. Suckling infants may be given a teaspoonful of sugar-water immediately before suckling, or immediately after. When a year old fine oatmeal is a useful article of diet, and the quantity of fatty food may be increased by giving butter with the oatmeal. Children a little older will often take greedily

[1] As to the connection between congenital constipation and hypertrophy of the colon, see an excellent article by Marfan (*Rév. des Mal. de l' Enf.*, 1895, t. xiii., p. 153).

butter, bacon fat, and even lumps of cold mutton fat. At about the age of one year, or earlier in a robust infant, broth, made with a little veal or chicken and vegetables, may be given, or the potato-milk recommended by Barlow (see p. 246). Young children will also take oranges and other fresh fruit, if properly prepared for them, at an earlier age than is generally proposed. Infants and young children often suffer from thirst, which is relieved better by lemon water made by adding fresh lemon juice to boiled water than by giving milk in any form. Milk should be regarded as a food and not as a drink. Such regulation of the diet, with the occasional exhibition of a small dose of compound liquorice powder, will generally do all that is necessary, and laxative and purgative drugs should as much as possible be avoided. When the habitual use of a laxative is thought to be desirable cascara sagrada will generally be found the least unsatisfactory. Small enemas of glycerine (5j–ij with about half the quantity of water) are useful in most cases of obstinate constipation with hard stools, and are probably the best routine treatment for cases in which the constipation is believed to be of the anatomical variety. If the sigmoid flexure, however, be full of fæces a large enema must be given and repeated until the whole mass has been passed. A glycerine injection should then be given daily, and as a rule, after a few months the action of the bowels becomes more regular, and in time the disproportion between the sigmoid flexure and the rest of the large intestine ceases, with the growth of the parts, to be a source of discomfort.

[The essential point in the treatment of constipation is to remember that it is after all only a symptom and that we must seek to determine the underlying cause of the symptom. In early infancy it is almost a normal condition, later it is abnormal and should be corrected by treatment of the patient, not of the symptom alone. In nurslings it is apt to be the result of constipation in the mother and it is the latter, not the baby, who should be treated. Analysis of the breast-milk should be made. Attention to the diet in bottle-fed infants and older children will generally correct the condition in these two classes of patients. Fruits are of value, and orange juice and prune juice may be given to an infant of a year. Older children may have a greater variety of fruit: baked apple, apple sauce, ripe pears, peaches, or plums. Massage of the abdomen, following the course of the colon, often helps to overcome the habit. This should be practiced at a definite hour each day.]

Prolapsus ani is a common complication of catarrhal inflammation of the large gut and of the rectum. It is most apt to occur in ill-nourished children, and is produced by straining at stool, or by straining in micturition, due either to phimosis or occasionally to stone in the bladder. At first there is merely a pouting of the rectal

mucous membrane through the anus at each motion. This gradually increases, or perhaps a sudden increase is produced by more than usually severe straining, and a large sausage-shaped mass, which bleeds easily, is found protruding from the anus. In rare cases the prolapse is due to rectal polypus which is grasped by the sphincter at each motion. As a rule in prolapse the sphincter is relaxed, and the prolapsed gut returns spontaneously or is easily reduced by gentle pressure. In some cases, however, there is considerable constriction at the anus, and the prolapse becomes deeply congested, oozes blood freely, and is reduced with some difficulty.

The rectum is invaginated through the anus, and to effect its reduction it must be grasped at its lower part, and squeezed gently upwards. To prevent recurrence the child should be caused to use a seat with a small aperture when passing a motion, and the thighs should be tied together, the buttocks and perineum being afterwards well washed. If the patient is an infant the nurse should be instructed to hold the thighs in contact when a motion is being passed. If stone in the bladder be present, or phimosis, they must be treated by ordinary surgical methods. Astringent injections into the rectum are seldom attended by more than very temporary improvement, and I have never found it necessary to resort to the subcutaneous injections of ergotine and strychnine in the neighborhood of the anus which have been recommended by some writers. In the great majority of cases the condition is merely a complication of ileo-colitis, and disappears when this is ameliorated by suitable treatment. Before reduction the prolapsed gut should be greased with ointment of galls or, if there be pruritus, with carbolic acid ointment (1 in 40). Hamamelis also (℥xx–℥j) is a useful application.

CHAPTER XXXIII.

INTESTINAL OBSTRUCTION.

Congenital—Acquired—Symptoms—Diagnosis—Treatment.

Intestinal obstruction may be due to **congenital defects** of development.[1] Thus the small intestine may be narrowed at various points, for example in the duodenum at the point of insertion of the common bile and pancreatic ducts, at the point where the duodenum joins the jejunum, or at or a little above the ileo-cæcal valve. The large intestine may be narrowed by displacement of the sigmoid flexure (see above), by a defect of development of the colon, or by narrowing of the point of insertion of the colon into the sigmoid flexure. Finally, there may be atresia ani, or imperforate anus. **Acquired obstruction** may be due to a variety of causes, which may be classified according as to whether they operate from within or without the gut. Thus the lumen may be contracted or completely obstructed by impaction of fæcal masses or foreign bodies, by new growths or by cicatricial contraction secondary to inflammation of the intestinal mucous membrane. Causes operating from without the intestine are false ligaments (bands) and adhesions, stricture due to protrusion through the ring formed by attachment of Meckel's diverticulum to the abdominal wall or mesentery, volvulus, intussusception, knuckling of the intestines, and the pressure of a tumour.

Of the congenital conditions imperforate anus is the most common; of the acquired, intussusception and the impaction of masses of hardened fæces or collections of foreign bodies.

The **symptoms** of intestinal obstruction are colic, vomiting, collapse, tympanites, and constipation. Each calls for separate consideration; all may, and probably will, develop sooner or later; the mode in which they develop may afford indications as to the nature and seat of the obstruction.

Colic, as a rule, comes on suddenly in the midst of perfect health, or after slight diarrhœa or constipation. The pain occurs in paroxysms, during which the form and vermicular motions of the gut may be plainly seen and felt through the abdominal walls. The intervals between the paroxysms lessen, until at last the pain is almost continuous. If the obstruction be in the small intestine, the pain

[1] See a paper by Monti (*Allg. Wien. med. Zeit.*, 1894, Nos. 35 and 36).

appears to start from the navel, and to radiate towards the stomach; if in the large intestine, the pain is referred to the regions occupied by the colon.

Vomiting, which is generally preceded by eructations, comes on soon after the colic sets in, and each attack of colic may end in vomiting. If the obstruction is very sudden and complete, the vomiting may be the first symptom, and is the more violent the higher up the obstruction. The vomited matters are at first, in all cases, the contents of the stomach, then glairy, bile-stained fluid, which after a time has an offensive odor. When the obstruction is in or below the middle part of the ileum the vomited matter eventually acquires a feculent odor, and with obstruction at or not far above the ileo-cæcal valve, actual formed fæces may be vomited.

Collapse is produced by the pain and vomiting. The face becomes pale, the eyes sunken, and there is a cold sweat on forehead and feet, small rapid pulse and hurried respiration. A condition of great mental depression almost amounting to unconsciousness ensues. At first the child revives a little between the attacks, but later the collapse is continuous.

Tympanites begins soon after colic and vomiting. Speaking generally, it is the greater and more extensive the lower down the obstruction. When this is in the rectum, sigmoid flexure, or descending colon, the distension affects first the colon, and tympanites is observed in the flanks and epigastrium; later the distension of the abdomen becomes general. When the lower part of the ileum, or the cæcum, is the seat of obstruction, the small intestine becomes distended, causing tympanites in the umbilical and hypogastric regions. Later the increasing distension of the small intestine causes the colon to be pushed away, and the tympanites becomes general. When the obstruction is as high as the jejunum, tympanites, if it is produced at all, appears late and is limited to the epigastrium; it may vary in degree, diminishing after vomiting. If in any situation the obstruction is incomplete, the distension is less and may diminish notably from time to time in connection with passage of flatus by the rectum.

Constipation, which develops sooner or later in acute obstruction, may be preceded by one or more actions of the bowels, which are either spontaneous or induced by enema. When the obstruction is high up the stools may even be copious; but eventually in these cases, and from an early stage in those in which the obstruction is low down, there is complete constipation, not even gas being passed. There may be, however, in association with the colic, severe tenesmus resulting in the passage of a little blood-stained or discolored mucus. This is particularly the case in obstruction of the colon, sigmoid flexure, or rectum. If the obstruction be not complete after several

days of constipation and tympanites, copious, loose, foul-smelling motions may be passed with great relief to the tympanites and all the general symptoms. In partial obstruction of the rectum hard scybala are passed with much painful tenesmus. In annular constriction of the rectum, or the lower part of the colon, the fæces may be of the thinness of a pipe stem, or flat and ribbon-like, or small like sheep droppings.

The *urine* is diminished in quantity when the obstruction is situated in the small intestine, and when very high up there may be complete suppression.

Palpation of the abdomen will reveal the existence of a tumour, or of an area over which there is an undue fulness in cases of obstruction by masses of impacted fæces, and in intussusception it is usually possible to feel a tumour, more or less sausage-shaped, in the left flank, or perhaps more towards the middle line. In intussusception, in addition to the ordinary signs of acute obstruction, there is commonly also a good deal of tenesmus, and frequent discharge of blood-stained mucus from the rectum. In all cases of doubt it is imperative to make an examination under an anæsthetic, when, as a rule, the tumour of intussusception is found to be well defined. Examination by the rectum should be practised in all cases of obstruction in which its seat and nature cannot otherwise be ascertained.

	Duodenum or Jejunum.	Lower Ileum.	Lower Colon.
Colic	Severe, radiating from navel to stomach.	Severe, radiating from the ileo-cæcal region.	Radiating pain over whole abdomen from left flank.
Vomiting	Early, violent bile-stained, and later foul-smelling.	With each paroxysm of colic; at first food, finally feculent.	Late, and only after paroxysms of colic as a rule.
Collapse.....	Early and severe.	Early.	Late.
Tympanites ..	Slight; epigastric; disappears after vomiting.	Marked; at first mainly umbilical and hypogastric regions.	Marked; limited at first to the regions of the colon.
Motions	May be copious at first.	May be copious at first.	Early complete constipation.
Palpation		If due to intussusception, cylindrical tumor.	
Urine	Early complete suppression.	Suppression may occur late.	Unaffected

The table above, compiled from Monti, may be of use in arriving at a diagnosis of the probable seat of the obstruction in obscure cases.

With regard to the **treatment** of obstruction no general rules can be laid down, owing to the great variety of lesions to which the condition

may be due. It is then inadvisable to give opiates until a diagnosis is made, as they are apt to mask the symptoms and cause the loss of valuable time (see appendicular peritonitis). Purgatives should not be given, but the large bowel should be irrigated by copious injections of boiled water. The stomach should be washed out, especially if vomiting be troublesome; this procedure nearly always gives relief, and in some cases has been followed by disappearance of the obstruction and complete recovery. Tympanites, at least, is almost invariably diminished by this means; but hot fomentations, with or without turpentine, should also be used for its relief. In intussusception the success of any treatment short of laparotomy must, to a large extent, depend on whether the two surfaces of the intussusceptum and intussuscipiens are or are not fixed by lymph. As the rate at which the lymph is effused differs very greatly in different cases, the duration of the case is not an infallible guide, though it is very improbable that the bowel will be unfolded except in a very early stage, since the intussusceptum soon becomes much swollen from obstruction to the circulation in its walls. Occasionally inversion, with massage of the tumour through the abdominal walls, has been successful; but such manipulations must be very gently performed. Distension with fluid (warm water or oil) or gas (from a gasogene or siphon) has succeeded. The quantity of fluid required will be from one to two pints. It must be injected slowly, and should be not cooler than 85° F. Care should be taken to see that the tube introduced into the rectum is passed without injury to the rectal wall, for the intestine has more than once been perforated and the fluid thrown into the peritoneal cavity. Failing reduction by these means, immediate laparotomy offers the best hope of recovery. If the reduction does occur, the patient should be kept at rest and given small doses of opium, which should be combined with belladonna.

CHAPTER XXXIV.

INTESTINAL PARASITES.

Tænia Solium—Tænia Mediocanellata—Bothriocephalus Latus—Tænia Canina; Symptoms of Tape-worm; Prophylaxis; Treatment—Ascaris Lumbricoides; Treatment—Oxyuris Vermicularis; Treatment.

Infestation by animal parasites is comparatively rare in infancy, but becomes progressively more common as childhood advances.

Tæniæ.—*Tænia solium* and *Tænia mediocanellata* are not uncommon in children over two years old, but are much less often met with under that age.

Tænia solium is a parasite of pigs. The fertilized egg swallowed by this animal undergoes development, penetrates the mucous membrane, and traverses the tissues until it reaches a muscle, generally that of the tongue, neck, or shoulder, where it becomes fixed in the intermuscular tissue, and there passes its cystic stage. It is hence known as the *cysticercus cellulosæ*. The cysts are about the size of a pea. In its cystic stage the worm may be a parasite of man also, sometimes in great numbers. It occurs in the intermuscular connective tissue, in the subcutaneous tissue, the eye, and the brain, where the symptoms it produces are those of cerebral tumor. If the living cysticercus reaches the human stomach with uncooked or insufficiently cooked pork, it undergoes development into the *tænia solium*. The scolex consists of two parts—the head and neck. The head is globular, and has at the summit a rostrum, or proboscis, surrounded by four suckers. The rostrum bears two concentric crowns of hooks. The neck is thin and has transverse striæ towards the lower extremity. As the animal grows, a series of segments form. They are at first broader than long, but become longer as they grow, until finally they are twice as long as they are broad. The segments are hermaphrodite, but after the fertilization of the ova in the uterus all the other organs atrophy, so that the ripe segment contains little more than the uterus distended by eggs. The genital sinus opens at the side by a pore, which is on different sides on alternate segments. The eggs are round and about $\frac{1}{170}$ inch in diameter. The ripe segment becomes detached, and it is by the observation of one or more such segments in the stools that the existence of the parasite in the intestines is first discovered by the patient. Before this

occurs the whole worm may have attained a great length, forming a long riband, whence its popular name tape-worm. The eggs contained in fæces reach the pig either in its food or by water. In cases of cysticercus in the human species, the eggs have probably entered the stomach with impure water. The *Tænia solium* is the most common tape-worm of man, and is not infrequent in children.

The intermediate host of **Tænia mediocanellata** is the ox. The egg, which is oval, reaches the animal either through food or water contaminated by human excrement, and becomes encysted in the muscles and viscera. It is killed by a temperature of 118° F., and can thus only reach the human intestine alive when beef is eaten raw or very imperfectly cooked. It grows there in the same way as the *tænia solium*, and may attain an enormous length. The head is flattened above, square rather than globular, and larger than that of *tænia solium;* it has four suckers, but no hooks. The segments when ripe are two or three times as long as they are broad; they are easily detached, and are passed frequently in chains of three or four. The genital pore has the same position as in *tænia solium*. This worm is said to be common in Abyssinia, and is attributed to the eating of raw meat. The practice of giving grated raw meat in infantile diarrhœa and in phthisis is believed to be increasing its prevalence in Europe. According to Osler, it is the commonest tape-worm in America.

Bothriocephalus latus.—The eggs of this worm are elliptical, possess an operculum, and are larger than those of the tæniæ. When free in water the egg develops slowly into a motile embryo, which can survive in this stage for some days or weeks. Certain fresh water fishes, especially the pike, serve as intermediate hosts, the bothriocephalus being found in the peritoneum and muscles. The worm, fully developed in the human intestine, has a relatively large almond-shaped head, without a rostrum or suckers, but with two lateral depressions. The neck is thick and flat. The fully developed segments are very large, and may be an inch long and nearly as broad. The genital pore is in the middle line and towards the front, but the eggs are extruded by another orifice farther back. This worm is very common in fishing villages in the Baltic, but is met with occasionally elsewhere.

Tænia canina (*cucumerina*) is a parasite of dogs. It has been met with occasionally in children, hardly ever in adults. It is a small, short worm; the head has a rostrum bearing four rows of hooks. The intermediate host is the dog-louse (*trichodectes canis*), and it occurs also, it is said, in fleas on dogs. The dog becomes infected by biting the itching parts and so swallowing the lice which contain the cysticerci. Children probably become infested by handling and kissing dogs and cats, and being "kissed"—*i. e.*, licked—

by dogs. *Tænia elliptica*, a parasite of cats, is probably identical.

Tænia nana is a small tape-worm very rarely met with. Billharz found a large number in the duodenum of a boy in Egypt. The head has a rostrum carrying a circle of hooks and four suckers.

The *symptoms* produced by the presence of tape-worms (*t. solium* and *t. mediocanellata*) are very indefinite. Dyspepsia, nausea, abdominal discomfort, diarrhœa, pruritus ani, and itching of the nose may be present; but these are common symptoms, and cannot with certainty be attributed to the presence of the worm or worms. In many cases no complaint of any kind is made until after the existence of the tænia is made known by the passage of the segments, either with the stools or separately. The children are generally thin and anæmic, and bothriocephalus can produce severe anæmia which may even be fatal. In a few cases jaundice has been observed as a complication, and has been attributed to the head of the worm being implanted near the orifice of the bile duct. Complaint is often made of headache, and vertigo and a large number of nervous symptoms have been attributed to the presence of tape-worm. Among these the most important are epileptic attacks, characterized by a long aura, a long convulsive stage (ten to fifteen minutes), and by a subsequent period of drowsiness or unconsciousness, which is also long. Such cases are very rare, and the connection between the fits and the presence of tæniæ is not well established. The same remarks apply with even greater force to chorea, mania, strabismus, amaurosis, and limitation of the visual fields, which, it has been asserted, may be produced by tape-worms.

The **diagnosis** depends entirely on the discovery of the segments passed from the bowel.

The **prophylaxis** is important. Measly pork or beef should not be used, though thorough cooking will kill the parasite. The use of grated raw beef may be a source of infection if the meat be not carefully selected, but the risk may be avoided by using chicken for this purpose. The segments, when passed, should be burnt.

In the **treatment of tænia** the patient should have a mild saline laxative, and should always take a very light diet—broths and soups, with little milk for two days, or at least for one day before the vermifuge is given. If the child has mucous diarrhœa, this should first be treated. The most trustworthy remedy is male fern. A child of ten may take ʒj of the liquid extract made up with peppermint water or other aromatic. It is advisable to give as large a dose as appears permissible on the first occasion, as it is believed that the drug on subsequent administrations has less effect than on the first. It should be given the first thing in the morning, fasting, and followed in about two hours by a full dose (half an ounce) of castor oil. In weakly children the dose of male fern may be smaller, but

very small doses are practically useless. In the more robust not only should the dose of male fern be larger but castor oil may be replaced by a calomel and jalap powder. Pomegranate root is also an efficient remedy. Half an ounce of the bark is macerated in two or three ounces of water and evaporated to an ounce, which may be taken by a child of five in three doses during the morning. Its alkaloid pelletierine is not to be recommended for young children, but at the age of ten or twelve either the sulphate or tannate may be given (gr. ij fasting). Turpentine has often been used with effect, with or without the addition of kamala. Osler gives a combination of pomegranate, pumpkin seeds, and male fern.

Ascaris lumbricoides, popularly called the round worm, is a cylindrical worm of a creamy or greyish red color. The female, which is four or five times as numerous as the male, measures some 8 to 12 inches, and is about the thickness of an ordinary lead pencil. Both extremities are pointed, the anterior more than the posterior. In the male, which is of about half the size of the female, the posterior extremity is curved into a hook. The body is marked by transverse lines. The mouth is at the anterior extremity, star-shaped, and provided with three chitinous nodules. The eggs, which are very numerous, are elliptical, of a brown color, measure .075 mm. by .058 mm., and have a double shell with an albuminous coating. They may be contained in the fæces in large numbers, and can withstand drying and freezing. In water they develop into embryos. There is no intermediate host, but the human intestine is infested directly by the eggs, which are no doubt carried by water, in which they can survive for some time.

This parasite is extremely common in children in temperate climates, but is still more frequent in tropical countries. It is more common in the country than in towns, and is said to be especially frequent in idiots and in children who have acquired the habit of eating dirt. It does not occur in infants nourished exclusively at the breast, and it is rare even in those fed artificially.

When the intestines are infested they usually contain more than one individual. The most common habitat is the lower part of the small intestine. The development of the worm is probably very rapid after it has become established in the intestine. When full grown it seeks to escape. As a rule, it finds exit by the anus, either with the stool or independently, being found not infrequently curled up in the bed, having escaped while the child slept. It may occur also in the colon, the duodenum, the stomach, and not very infrequently escapes by the mouth, either with or without vomiting. It may find its way into the naso-pharynx, and may thence be extracted by the child. In rare cases it has been known to enter the Eustachian canal, and appear at the auditory meatus; it has become

impacted in the larynx, causing sudden death, and has reached a bronchus and led to gangrene of the lungs. The worm may also force its way into the bide ducts, causing jaundice, dilatation of the bile ducts, and suppuration. Osler mentions a case in which the common duct and the main branches thoughout the liver were enormously distended and packed with numerous worms. In ulceration of the intestine in enteric fever or tuberculosis, the worm may pass through into the peritoneum ; but the assertion that it can pass through the healthy intestinal wall, and so lead to peritoneal abscess, cannot be accepted. It has also found its way into the bladder. When very numerous the worms may become rolled together, forming large masses which have caused intestinal obstruction ; otherwise the ascaris does not produce any lesion of the intestine. Abscesses containing one or more worms have been met with in the inguinal and umbilical regions.

The number of **symptoms** which have been attributed to the presence of round worms in the intestine is legion. While it is certain on the one hand that even an immense number of ascarides may be present—Massini has recorded the case of a girl aged three years, who during less than two months passed 3,000—without any symptoms whatever, yet it will be found that children infected with these worms often present various signs of ill-health—restlessness, colic, picking at the nose, anal irritation, pallor, and dark rings round the eyes. Further, in neurotic children various nervous symptoms may be dependent on the presence of round worms, and disappear when they have been expelled. Headache, and choreiform movements, and in younger children convulsions and symptoms suggesting meningitis (retraction of the head, vomiting, loss of consciousness, and dilatation of the pupils) may disappear after one or more round worms have been passed. When numerous, marked anæmia may be produced, and Demme[1] has recorded cases in which the symptoms resembled those of pernicious anæmia. In one of these cases the number of red blood-corpuscles was 2,450,000, the proportion of white to red corpuscles 1: 90, and the hæmoglobin 40 per cent. After the passage of a large number of round worms the number of red red blood-corpuscles rose in a few weeks to 4,100,000 the hæmoglobin to 70 per cent. and the proportion of white to red corpuscles fell to 1: 160.

The **diagnosis** must rest on the passage of a round worm, or on the discovery of its eggs in the fæces. In doubtful cases a vermifuge should be administered.

In the **treatment** the most reliable drug is santonin. It causes the worm to become detached, and should be combined with small doses of calomel, or followed by castor oil or a saline laxative to ensure

[1] *Jahrb. f. Kinderhlkd.*, Bd. xxxv., S. 276.

expulsion. The dose for a child of three should be 1 to 1½ grains. This should be given at night, and the laxative in the morning. If the daily dose be divided into three, given at intervals of one to two hours, beginning in the early morning, the effect is perhaps better. The main objection to its use is that it sometimes produces nausea, yellow vision, urticaria, or a scarlatiniform erythema, and some pain on micturition. A very large dose may produce much more serious symptoms; vomiting, dilatation of the pupil, dyspnœa and cyanosis, convulsions, epistaxis, and hæmoglobinuria. The child may become collapsed, and death has been known to occur. These symptoms, which have been observed usually after rather indiscriminate administration of "worm tablets" by the parents, are possibly due to some impurity in the drug; but it is advisable to begin with small doses, and after the third morning to stop the drug for a week or ten days, then to give a dose of calomel with jalap or jalapine, and resume the santonin if the worm or its egg are found in the stools. Oil of turpentine is also an effectual remedy. The dose should be ʒj for a child of ten, prescribed with mucilage of tragacanth in infusion of senna.

Oxyuris vermicularis.—The common thread worm or seat worm inhabits the colon and rectum, but conjugation takes place probably in the lower part of the ileum. The female is about ⅓ inch long (9 to 10 mm.). It occurs in much larger numbers than the male, which is about half its length. The end of the tail in the female is sharp, in the male blunt and furnished with a spiculum. The eggs, which are 3 to 5 μ in diameter, are swallowed with water, salads, etc., but reinfection is possible, the eggs being carried from the anus to the mouth by the fingers. Catarrh of the lower bowel favors the establishment of the parasite. The parasites wander at night to the anus, by which they escape, causing great itching and irritation. Children, especially between the ages of two and five, are particularly prone to be the hosts of these worms, which may, however, be present at any age.

The **diagnosis** may be made by the observation of the worm. It may be found in the folds of the anus, or its eggs may be found in the fæces. It has occasionally been passed through the mouth.

The **symptoms** are mainly those of local irritation, intense itching and burning, coming on usually at night, and waking the child up. It scratches the anal region and rubs its thighs together; in this way is produced an eczematous condition about the anus, and intertrigo in the inguinal region, where, indeed, the worm may be found occasionally. In girls the parasite may wander into the genital passages, causing great itching and irritation. In both sexes, but especially in the female, it is believed to predispose to nocturnal incontinence of urine. The effect of the presence of these parasites

on the general health may be very injurious, owing to loss of sleep and constant irritation. The child is thus predisposed to nervous disorders, though it is doubtful whether either convulsions or chorea can be directly attributed to their presence.

Treatment must be directed: (1) To the relief of the itching by carbolic, belladonna, or weak white precipitate ointments made with vaseline, and by the injection of a small quantity of olive oil (℥ j–ij) into the rectum. (2) To clearing the rectum and sigmoid of the worms, for which purpose larger injections of common salt (℥ j in ℥ ss of cold water), infusion of quassia, carbolic acid solution (1 in 100), vinegar and water (equal parts), or turpentine (℥ j–ij mixed thoroughly with soapy water) may be used once a day. (3) To destroying the parasites in the ileum and upper colon, where probably is their breeding-place. A laxative should be given, either a saline, or the old-fashioned compound rhubarb, or compound liquorice powder, followed by vermifuges. Santonin is often used for this purpose, but a continuous action is to be sought, and it is not desirable to continue the use of this drug for more than three or four days. Very good results may be obtained with naphthalin, given four or five times a day for two days after the bowels have been well opened, then suspended for a week, and repeated if necessary for two days more. A third and a fourth course may be necessary. The dose for a child of two is gr. ij–iij in powder with sugar. Naphthaline is soluble in fats, and they should be excluded from the diet during the treatment, as the solution not only interferes with the action on the oxyuris but may lead to symptoms of general poisoning. (4) To improve the condition of the lower bowel, which is usually affected by catarrh. (5) To prevent reinfection. The child's room and all clothing, bedding and toys should be scrubbed and disinfected. The finger-nails should be kept short and well washed. Frequent bathing and change of linen are advisable.

CHAPTER XXXV.

HYDATID DISEASE.

Tænia Echinococcus—Geographical Distribution—Hydatid of the Liver; of the Lung; of the Heart; Intracranial; of the Kidney and Spleen.

Tænia echinococcus is a very minute tape-worm which infests the dog. It consists of a head and three segments, only the last of which is mature. The whole measures about $\frac{1}{6}$ inch in length. The ripe segment may contain as many as 5,000 eggs, which are ovoid, about 0.01 mm. in long diameter and provided with a shell. Swallowed with water, or on herbs, by the ox, pig, sheep, or man the shell is dissolved, and the embryo, which has six hooks, burrows through the walls of the gastro-intestinal canal and may reach the peritoneal cavity or the muscles. It may enter a blood-vessel and be then carried to various organs, especially by the portal blood to the liver. Once arrested in an internal organ the embryo passes into the cystic stage. The peculiar characteristic of this parasite, and that which gives it its clinical importance, is that instead of remaining single, and in size small, it multiplies by a process of internal gemmation, so that the much distended primary cyst may eventually contain a number of included or daughter cysts, and these daughter cysts, again, a further set of grand-daughter cysts. From the lining membrane of the cysts buds arise and develop into scolices, which are, in fact, the heads of *tænia echinococcus*. When swallowed by the dog they develop into that tænia and attain sexual maturity. The budding begins about five months after the embryo has become fixed. The cyst can continue to live and to grow for years. If the parasite die, the contents of the cysts undergo inspissation, and are converted into a granular, putty-like material. The fluid contained in a living hydatid is clear and colorless. It contains no albumen recognizable by ordinary clinical tests, but about 0.5 per cent. of chloride of sodium, traces of succinates and sugar, and a toxic body, probably of the nature of an albumose. The hydatid cyst becomes surrounded except in the lungs by a capsule of fibrous tissue derived from the organ in which it is imbedded. Suppuration may occur within this cavity, and may lead to rupture and to grave symptoms. Rupture may also occur without suppuration, either spontaneously or as the consequence of a blow.

The **geographical distribution** is peculiar. It is certainly very uncommon in Great Britain and on the continent of Europe, especially in children, and appears to be unknown among native-born Americans. In Australia, however, it is very common, and children seem to suffer, at least, as much as adults. In the Children's Hospital, Sydney, N. S. W., 1 case out of 173 admitted suffered from hydatid (Stirling). This is almost the same proportion as that for all hospital patients in New South Wales, which, according to Stirling,[1] is 1 to 175. Thomas found that of the fatal cases which he collected[2] 7.6 per cent. occurred under the age of ten years, and 10.7 per cent. between the ages of ten and twenty. As he points out, a suckling child is very unlikely to receive infection, and the youngest patient that he had met with was a child aged two years and one month upon whom he operated for hydatid of the liver.

As in adults, the most usual site is the **liver.** Of 120 cases of hepatic hydatid collected by Murchison, and in which the age is stated,[3] 12 were in children under ten years, and 16 between ten and twenty years. The symptoms and physical signs are those of a slowly growing tumor of the liver. It is usually painless, though not always, for complaint of aching pain in the liver is made not infrequently before definite swelling can be detected. It is elastic or fluctuating, and may afford hydatid fremitus. The physical signs of hydatid of the convexity resemble those of hydatid of the base of the lung, and it may be impossible to distinguish between these two conditions. Further, in both cases the physical signs are much the same as those of a limited effusion into the pleural cavity. This point is discussed below. Hydatid, moreover, may produce pleurisy. Hydatid of the lower surface of the liver may be mistaken for hydronephrosis, but it is usually more superficial than a renal tumor, and pushes down the colon in front of it. Frequently it can be felt as a rounded swelling giving an irregular outline to the lower edge of the liver. It moves with respiration, and on percussion is continuous with the liver dulness. Hydatid of the liver may rupture into the stomach or intestines, the bronchial tubes, the pleural cavity, or the peritoneum, through the abdominal walls, or into the urinary passages, the order of frequency being that here given. Of these accidents, rupture into the intestine appears to be most favorable to the patient. Rupture into the lungs may cause sudden death from suffocation or, if that is escaped, long and exhausting illness with the symptoms of basic cavity or pulmonary gangrene. Rupture into the pleural cavity causes empyema, and usually leads

[1] *Aust. Med. Gaz.*, August 20, 1895. He gives the proportion for all Australia as 1 to 206, at all ages.

[2] "Hydatid Disease, with Special Reference to its Prevalence in Australia," by John Davies Thomas, M.D. (Edited by Dr. Lendon), 1894, vol. i., p. 126.

[3] "Clinical Lectures on Diseases of the Liver," Sec. Ed., 1885.

to a fatal result unless operated on. Suppuration of the sac may occur spontaneously ; but it is found to occur with great frequency as an after result, immediate or later in life when resort has been made to tapping. The constitutional disturbance produced by suppuration is grave, and is characterized by pyrexia, rigors, rapid pulse, and emaciation. In the treatment of hepatic hydatid the immediate dangers of puncture and aspiration, even with the strictest aseptic precautions, are great.

At a later date suppuration in the sac very frequently occurs and requires the radical operation. Thus the large experience gained in Australia [1] thoroughly supports the reasons and statistics advanced by Thomas for the conclusion that the safest method of **treatment** is by abdominal section, immediate removal of the mother cyst, and stitching the sac to the parietal wound (Lindemann's operation). Further, experience shows that in simple cysts that have not suppurated the intraperitoneal method gives the best results.[2]

Hydatid of the lung is, in Australia, by no means rare in children. The **symptoms** do not differ from those observed in the adult, the most prominent being a dry cough with little or no expectoration, but with occasionally slight hæmoptysis [3] and dyspnœa on exertion. Complaint of pain in the chest is not made unless the cyst be large. Slight hæmoptysis before rupture proceeds from the congested lung surrounding the sac. It may be profuse after rupture or incision. Pyrexia and emaciation are not marked unless the cyst has suppurated.

The **physical signs** produced by a hydatid of medium size near the surface, at or towards the base, to some extent resemble those of a localized pleural effusion. Together with deficient expansion there is an area, tolerably sharply defined, of absolute dullness, with a sense of increased resistance on percussion. With these signs there is absence of respiratory murmur, vocal fremitus, and resonance in the same region. Above, there is increase of respiratory murmur and of resonance on percussion. If the cyst be large, there is bulging of the chest wall, with distension of the intercostal spaces, where fluctuation can frequently be obtained. The heart, liver, or spleen may be displaced.

The **diagnosis**, often difficult (unless there be no general grounds or reason to suspect hydatid, which is not the case in Great Britain or America), must depend partly on the complete absence of vocal resonance, but mainly on the general condition of the patient, who will be free from emaciation or fever, and upon the prolonged history.

[1] *Trans. Aust. Med. Congress*, 1892 ; *Aust. Med. Gaz.*, 1895.

[2] Bond, *Brit. Med. Journ.*, 1891, vol. i., p. 795. Conf. *Intercolonial Quarterly Journal*, February, 1895.

[3] Graham states ("Hydatid Disease," 1891) that in some cases hæmoptysis may not occur.

If the cyst be deeply situated, there may be much difficulty in recognizing its existence. The compression of the lung tissue produces a high-pitched or tympanitic note on percussion. The physical signs of hydatid cyst at the apex resemble those of early phthisical consolidation. The diagnosis must depend on the absence of breath-sounds. If the cyst ruptures into a bronchus, an acute suffocative attack occurs, and the lungs are flooded with fluid, which is coughed up in large quantities. Later, shreds of the cyst wall or daughter cysts are brought up after severe cough with suffocative attacks. The physical signs change, the patient suffers from fever and night sweats, and the profuse purulent expectoration contains sloughing particles. In time clubbing of the fingers is produced. Owing to the fluid contents of the cyst being replaced by air, the physical signs become those of pulmonary cavity, or, if a communication is established with the pleura, of pneumothorax. The diagnosis must rest mainly on the history, on the recognition of any fragments of the cyst wall which may be coughed up, on the general condition of the patient, and on the fact that in children large cavities are very rare. The absence of the bacillus tuberculosis from the sputa will afford confirmatory evidence.

The **treatment** of pulmonary hydatids must be surgical. Thomas expresses the opinion that the probability that any case in which pulmonary hydatid can be diagnosed will undergo spontaneous cure is virtually *nil*. The best treatment appears to be free opening into the sac, and the immediate removal of the parasite. Puncture of hydatids of the lung, from the dangers immediate and remote, must be looked upon as a most hazardous proceeding.[1] Removal of the cyst is followed by re-expansion of the lung.[2]

Hydatid disease of the heart is a very rare event at any age, but an unusually large proportion of recorded cases have occurred in early life. Out of 33 cases collected by Thomas, in which the age is given, 4 occurred under ten years of age and 11 between eleven and twenty. In the majority of cases the parasite was imbedded in the muscular substance. The condition is not necessarily fatal, since the parasite may die, but the more usual termination is sudden death, which may be brought about by rupture into one of the cavities of the heart, generally into the right side.

A considerable number of cases of **intracranial hydatids** have been recorded in children. In 79 cases collected by Thomas[3] 19 per cent. occurred under ten years of age and 34 per cent. between eleven and twenty, so that more than half the cases occurred under twenty. The cerebral hemispheres are the most usual site. In the

[1] *Trans. Med. Congress Aust.*, 1892, pp. 381 and 441.
[2] Lendon, *Aust. Med. Gaz.*, 1895, p. 477.
[3] *Loc. cit.*, vol. ii., p. 86.

majority of cases there is a single cyst, which is generally of spherical or ovoid form. If a capsule be present it is usually fine and soft.

The **symptoms** of intracranial hydatids are those common to intracranial tumor. The general symptoms—headache, vomiting, and optic neuritis—may all be present, but in some cases they are not well marked, and the only symptom of which the patient complains is headache, occurring in paroxysms and aggravated by movement. Such cases may terminate suddenly, without any suspicion of the real cause of the headache being entertained. As will be gathered from what has been said, localizing symptoms may be absent. Epileptiform seizures, in some instances limited to one side of the body, have occurred, and in others definite hemiplegia, although this has been absent in cases in which from the size of the hydatid found after death it might have been expected to occur. In some cases the cranium has been enlarged, generally or locally, and in a few the bones have become perforated. Vertigo is not a constant symptom, and a staggering gait has been noticed chiefly in cases of cysts occupying either the posterior lobes of the cerebrum or the cerebellum. Blindness would appear to have been produced in an unusual proportion of the cases.

The **duration** of the disease is now well ascertained; but Thomas states that the average duration of life (at all ages) after the appearance of the first cerebral symptoms is about one year. The diagnosis, even in those cases in which well-marked symptoms of intracranial tumor arise, must generally be in the main conjectural. If the symptoms point to cerebellar tumor, it is extremely unlikely to be hydatid; but in chronic cerebral tumor the possibility of its being a hydatid cyst should be borne in mind, and the probability would be increased if there is evidence of localized thinning of the cranial bones.

The **treatment** of intracranial hydatid by drugs offers no prospect of improvement, and relief can only be afforded by surgical means.

Hydatid disease of the kidney or spleen is extremely rare. Thomas could find no recorded cases under ten years of age, though he mentions a few between the ages of eleven and twenty. Cases of hydatid disease of the spinal cord and superficial structures are among the curiosities of medical literature.

CHAPTER XXXVI.

DISEASES OF THE GENITO-URINARY SYSTEM.

The Urine — Albuminuria — Albuminuria of Puberty — Hæmaturia — Hæmoglobinuria—Pyuria—Diseases of the Kidney: Diffuse Nephritis; Glomerulo-Nephritis; Amyloid Degeneration; Uric Acid Diathesis; Renal Calculus; Hydronephrosis; Pyelitis; Perinephritic Abscess; Congenital Cystic Disease of the Kidneys; Tumours of the Kidney.

Urine.—The kidneys are relatively larger at birth than in the adult; the weight is to the total body weight as 1 to 120 instead of 1 to 240 as in the adult. The lobulation is marked; the lower end of the kidney reaches almost to the level of the iliac crest. The organs are active during fœtal life, and it is not uncommon for some urine to be passed immediately after birth. On the other hand, no urine may be passed for twenty-four hours, and there are great variations in the quantity. The urine first passed has a specific gravity of about 1010, and is often turbid from the presence of urates, epithelial cells and mucus. Uratic infarctions are very frequently present in the terminal straight tubules of the kidneys at birth, and are probably physiological. They consist of uric acid, ammonium urate, and amorphous urates, mixed with mucus and epithelial cells. They are washed away usually within a week or a fortnight, but may persist for seven or eight weeks; these uratic *débris* produce brickdust red stains on the napkins. In early infancy the urine is quite colorless or of a very pale primrose color (Chablis wine), and odorless, or nearly so. The specific gravity falls soon after birth, but rises again at the beginning of the third week to 1006 or more. It is difficult to collect the whole of the urine in infancy, as defæcation is usually accompanied by micturition; but the quantity passed daily shows considerable variations in different children and at different ages. It appears to increase rapidly at first, and by the third week is relatively more copious than in the adult; this excess persists during the suckling period. Specimens may be obtained by keeping a perfectly clean dry sponge in contact with the genitals, or by passing a catheter. After the third or fourth month a skilful nurse will usually be able to obtain the greater part of one or two micturitions during the day in a clean vessel. The quantity passed at each micturition does not vary much. This uniformity may be attributed in part to the uniformity of the diet, and in part to the fact that in

young infants micturition is a purely reflex act. The age at which some voluntary control is acquired varies much; some power of holding water may begin to be exercised soon after the sixth month, but even earlier than this the infant may acquire the power of micturating when solicited. A healthy infant of two to three months old may be expected to micturate about ten times in the twenty-four hours, and to pass altogether from 8 to 10 fluidounces; by six months the daily quantity will be doubled or trebled, so that before weaning as much as a pint and a half may be passed. After weaning, this quantity will decrease, and a child of two or three years may not pass more than a pint, or a pint and a quarter. The reaction of the urine in early infancy is neutral, or slightly acid, seldom alkaline. The quantity of urea in early infancy is relatively large; it increases as the infant grows, but not in proportion to its increase of weight. A notable decrease in the amount commonly occurs at weaning, and an attack of diarrhœa may cause a great diminution. The statements as to the proportion of uric acid present are conflicting, and it is probable that its amount depends a good deal upon the condition of nutrition and of the digestion. A trace of albumen may be discoverable during the first few weeks of life; probably it is to be attributed to the irritation produced by uratic infarcts, and is not of serious significance.

[The factor of excretion is such an important one and the well-being of the subject depends to such an extent upon its proper performance, that examination of the urine is of great importance. Much may be learned from such examination. We do not of course find actual, organic disease in all cases but we may determine to what extent metabolism is going on and in what respect it is deficient, and may thus gain valuable information in the matter of treatment.

Investigation of the urine of children has not been carried out as extensively as the importance of the subject demands. Normal physiological standards are necessary with which to compare the results obtained from the examination of pathological specimens, but comparatively few investigations have been made into the urine of healthy children, and well-established definite standards of urinary analysis in children at different ages have yet to be accurately determined. Many analyses from children of every year up to puberty must be made before these standards can be established; for the composition of the urine varies from year to year in childhood. The publication of analyses of pathological cases is also important, and from a study of such, much will undoubtedly be learned which will be of help in preventing the development of actual organic disease later in life. We must bear constantly in mind in the treatment of children that we are dealing with an organism that is plastic and still

[1] Longet quoted by Ballantyne, "Diseases of Infancy," 1891, p. 178.

in the formative period, with only tendencies, not actual habits established. There can be no doubt of the truth of Weissmann's theory that it is tendencies, not actual traits, which are transmitted from parent to offspring. The earlier we attempt to mould these tendencies, the more impression can we make upon them. The study of a large number of urinary analyses from children with known hereditary tendencies will undoubtedly show along what lines these children are apt to develop, and, if these lines be in the wrong direction, the first step towards correcting them has been taken. It is the reviser's belief that from careful, exhaustive study of the urine much may be learned of value in heading off serious organic disease of the kidney later in life.

The tables given below are compiled from a study of the urine of 70 healthy children ranging in age from 1 day to 12 years, 48 girls

Age, yrs.	Weight, klg.	Sex.	Total Specimens.	Amount, c.c.	Specific Gravity.	Urea, per cent.	Total Urea, grm.	Chlorides, per cent.	Phosphates, per cent.	Sulphates, per cent.	Amount to 1 klg. Body Weight, c.c.	Urea to 1 klg. Body Weight, grm.
3	16.8	2 M	5	358	1024.0	2.2	7.87	12.5	12.00	1.08	21.3	.468
4	16.9	7 M 4 F	31	299	1027.1	2.9	8.67	11.17	9.22	1.41	17.6	.513
5	16.8	1 M 7 F	17	392	1024.5	2.6	10.19	10.37	8.89	1.13	23.3	.606
6	16.7	2 F	2	405	1023.0	2.7	10.94	11.00	6.50	1.13	24.2	.655
7	20.9	1 M 3 F	4	564	1018.0	1.1	6.20	7.00	5.63	0.85	26.9	.296
8	22.6	2 M 7 F	20	628	1021.1	2.2	13.82	9.16	7.32	1.11	27.8	.611
9	26.2	1 M 9 F	25	731	1020.7	2.3	16.81	8.46	7.61	1.10	27.9	.641
10	27.5	2 M 6 F	15	768	1023.5	2.1	11.28	9.29	6.85	1.10	27.9	.592
11	27.2	1 M 3 F	7	716	1018.8	2.3	16.83	10.03	7.50	0.92	26.3	.618
12	36.4	2 M 3 F	8	829	1021.5	2.8	23.21	9.27	6.77	1.00	22.7	.637
Tot.		19 M 44 F	134									

and 22 boys. The results are classified as to age and averaged; 146 specimens were examined, it being better to examine two or more from a limited number of cases than a single specimen from a larger number of children. The results justified this method of procedure, as considerable variations in the amount of urine passed at different times were noticed.

Age.	Weight.	Sex.	Amount in Single Specimen, c.c.	24 Hour Amount, c.c.	Specific Gravity.	Urea, per cent.	Chlorides, per cent.	Phosphates, per cent.	Sulphates, per cent.	Remarks.
1 day	3.18 kil.	F	3			.4				
12 days	3.86 K.		36		1003	.1	Passed at 12 M.			
3 wks.	4.14 K.				1001	.5	" 9 A.M.			
$3\frac{1}{2}$ wks.	4.25		30		1005	.2	" 4 P.M.			1 hour after breast.
4 wks.	4.47	M	23		1004	.1				
1 year	9.98		9		1021	2.6				
1 year					1026	2.7	12	16	1.75	Dentition slow.
$1\frac{1}{2}$ years		F		195	1030	2.5	18	11	1.25	1 Specimen.
13 mos.	9.76 kil. ($21\frac{1}{2}$ lbs.)	F			1026	2.3	11	11	1.00	Mixture night and day urine.
20 mos.				290+	1028	2.8	16	13	1.75	
$20\frac{1}{2}$ mos.		M		325+	1026	2.1	8	12	1.00	
2 years	14.07 K. (31 lbs.)	M		450	1020	1.7	6.5	6	1	
		3 M, 3 F	12 specimens in all							

Observations were made upon the following points: Total amount of urine in twenty-four hours, color, reaction, specific gravity, presence or absence of albumen and sugar, percentage and total urea, percentages of the chlorides, phosphates, and sulphates. The sediment was examined macroscopically and microscopically. The ferrocyanide and nitric acid tests were employed for the albumen, the urea was estimated by the hypobromite method, Doremus' ureometer being used, the percentage of the chlorides, phosphates, and sulphates determined by centrifugal analysis as recommended by Purdy, the tubes being revolved three minutes at a speed of 1,100 revolutions per minute.

Examination of these results in detail show several interesting points:

Amount.—The daily amount is much less than that recorded by most authors, except Herz, whose analyses upon 60 cases, 30 girls and 30 boys, correspond approximately with the reviser's. Other observers, however, Schabanowa, Cruse, Camerer, Berti, Pollak and Martin-Ruge record larger amounts. The difficulties of collecting urine from active, healthy children are of course great, and the suspicion at once arises that all the urine in these cases was not saved; great care was taken in this respect, however, and no cases about which there was any doubt were included in these analyses. Furthermore the specific gravity confirms the amount.

Specific Gravity.—This, as we see, is comparatively high, a con-

dition we should expect in children passing but a small amount. Had the amount actually been larger, we should have expected a lower specific gravity.

The specific gravity of one young infant recorded is low, coinciding with the well-known observations at this period; it ranged from 1,001 to 1,005 from the 13th day to four weeks. It is, however, generally higher during the first two days of life before the establishment of the breast-milk. It drops after this and continues low throughout the first year, owing to the fluid character of the infant's food. During the second year, solid food being added to the diet, the specific gravity rises and in four cases, aged respectively twelve, thirteen, eighteen, and twenty months, was found ranging from 1,026 to 1,030, the urine being a mixture of the day and night elimination.

Urea.—The estimation of this constituent is perhaps the most important of all the urinary solids, being as it is an index of general metabolic activity. As we should expect from their greater activity, and as Purdy and Foster state, we find the urea excretion in children relatively higher than that in adults. The low percentage noted during early infancy is, of course, due to the quiescent state of the child. Martin-Ruge, however, reports wide variations in single specimens during the first ten days of life, ranging from 6 per cent. to 1.9 per cent. Schiff also gives wide variations, placing averages at from .28 per cent. to 1.7 per cent. during the first fourteen days. Why there should be such a wide range in the excretion of this substance at a time of such quiescence it is difficult to see. Possibly greater metabolic activity after nursing may account for it. I have no statistics upon the relative amount of urea in urine passed just before, just after, and some time after feeding. The few observations made at this age show without exception very low percentages, from .4 per cent. to 1.2 per cent., lower than those cited. After the first year it rises, and from three to twelve years, the 133 specimens show a higher general average than that usually given. Verordt's percentage based on seven cases is recorded at 1.1 per cent. to 2 per cent., four being below 2 per cent., one 2 per cent. and one 2.6 per cent. and one not given. This represents the adult average, whereas so great is the physical activity of the growing child, so active is his metabolism, that a large amount of urea is formed, and while it may be argued that most of his nitrogenous food goes to the building up of the rapidly growing body, and thus the amount of urea formed in the urine would naturally be less, it would seem more rational to expect a greater elimination of this substance. Not only are my average percentages higher than the average given for adults, but individual cases show a remarkably high percentage of elimination of urea, eight children having over

3 per cent., the highest being 3.7 per cent. The amount of urea per kilogram of body weight, while slightly higher than the ratio given for adults, is lower than that given by other observers, as we should expect from the smaller amount of urine.

Chlorides.—The chlorides were found quite constant at about 11 per cent. up to seven years, after which they were about 9 per cent.

Phosphates.—The phosphates were found to be from 8 to 11 per cent. from 3 to 5 years; 5 to 7 per cent. from 6 to 12 years, the adult range being about 8 per cent. It has been suggested that the smaller amount of phosphates found in the urine of children is due to the fact of the phosphoric acid being retained in the body for the growth of bone. One specimen from a year old boy showed 16 per cent. and as he was somewhat slow about teething, though otherwise perfectly healthy, the question suggested itself as to whether substances which normally go to build up the teeth, were being eliminated as phosphates, and if so, why; digestion was absolutely normal. No conclusions, however, can be drawn from one solitary instance; the observation is merely of speculative interest.

Sulphates.—The percentage of sulphates was 1 to 1.2 per cent., slightly higher than in adults, .8 per cent. being their average. Purdy states that the sulphates run parallel with the urea.

Albumen and Sugar.—Neither albumen nor sugar were detected in any specimen. So much had been said about a physiological albuminuria that I had expected to find albumen in one or more specimens. It must be remembered, however, that my cases were examined but two or three times, and some only once, and that therefore a transient temporary albuminuria might have come and gone between examinations.

Sediment.—Examinations of the sediment showed nothing of special interest in any case.

Reaction.—The reaction was acid in all cases, though of course varying in intensity in different specimens.

Color.—The color in most cases was pale, in the rest normal.

Looked at as a whole the records show three factors of chief importance: the small amount of urine, the high percentage of urea, and, a natural result of these two, a high specific gravity. In other words, these children were passing a comparatively concentrated urine. They were all healthy, robust children, eating, sleeping, and digesting well, and of average weight. They were average American-born children, living in an asylum under American customs and régime, though mostly of foreign parentage. The chief point of difference between these analyses and those made by foreign observers is the larger amount of urine recorded by the latter.

It is interesting to note in this connection the amount of urine in

adults, as given by observers in different countries. Simon gives the following:

Salkowsky (Germany)	1500 to 1700 c.c.
Jaksch (Austria)	1500 to 2000 c.c.
Landois & Sterling (England)	1000 to 1500 c.c.
Gautier (France)	1250 to 1300 c.c.
Simon (America)	1000 to 1300 c.c.
(Females)	900 to 1000 c.c.

It would seem from these tables that American adults, and also children, pass smaller amounts of urine than the people of other countries.

This factor of a concentrated urine, small in amount, occurring in children, can but be regarded as unfortunate, inasmuch as such urine is a source of constant irritation to the kidney and hence likely to pave the way for organic disease of that organ. Especially in those children with a lithæmic or rheumatic inheritance would such a condition if long continued be a serious matter. They, and indeed all children, should be encouraged to drink freely of milk and of water. This is all the more important when we consider the numerous conditions which directly and immediately, indirectly and remotely, act as causes of nephritis, a disease more common in early life than is generally supposed.]

By **albuminuria** is to be understood the presence in the urine of albumen, recognizable by the ordinary clinical tests: (1) Fuming nitric acid in the cold, and (2) boiling, with subsequent addition of an acid, preferably acetic acid.

Albuminuria is evidence of changes in the renal structures. The changes may be transient, and limited to the epithelium of the glomeruli and tubules.

Albuminuria of Puberty.—It is not uncommon in children of either sex, but most often in boys, to meet with cases in which, at about the time when the changes attending puberty are commencing, albumen appears in the urine in small quantities. Such patients are described as "delicate"; they are thin, have a poor appetite, are easily tired, and suffer often from headache, but they do not present anasarca. The albumen may be present only during part of the day (*cyclic albuminuria*), and will then usually disappear if the patient be kept in bed. The amount is increased by exertion. In some cases granular or hyaline casts may be present; whether this be so or not, it is safer to look upon such cases as being, in reality, examples of a very mild form of subacute or chronic nephritis, and the patient should be treated on the same principles as those followed in the management of convalescence from subacute nephritis. The prognosis is good if proper treatment can be instituted.

Hæmaturia.—Blood derived from the kidneys is mixed uniformly with the urine, to which it gives a smoky appearance, or, if in larger quantities, a bright red or deep porter color. A minute quantity does not alter the color of the blood and can be recognized only by microscopical examination. Clots may be derived from the pelvis of the kidney or from the bladder. Blood from the bladder may be mixed intimately with the urine or passed at the end of micturition; its commonest cause is stone.

Hæmaturia may be due to the effect on the kidneys of malignant attacks of the acute specific diseases, or of certain poisons (*e. g.*, cantharides, turpentine, carbolic acid) to acute glomerulo-nephritis (*e. g.*, post-scarlatinal nephritis), to congestion, as in heart disease, whether congenital or acquired, to renal infarction, to tubercle, to new growths, or in certain tropical and sub-tropical climates to the *filaria sanguinis hominis*, the bilharzia, or to malaria. Injury of the kidney, or of any part of the urinary passages, may lead to the appearance of blood in the urine. Calculus in the pelvis of the kidney is, in childhood, one of the commonest causes of hæmaturia not due to acute febrile disorders. The quantity of blood varies; it may be absent altogether for considerable periods, it may be present in microscopical quantity only or, again, in sufficient amount to alter the color of the urine (smoky or red). Transient recurrent hæmaturia may also be observed in cases in which the urine contains microscopical crystals of oxalates or uric acid. Occasionally blood, apparently derived from the kidneys, is present in the urine of children without any discoverable cause (renal epistaxis, Gull).

Hæmoglobinuria is a condition in which the urine contains free hæmoglobin or methæmoglobin. According to the quantity of either present, the urine has a smoky, lake, brown-red, or black color. It deposits a dark brown sediment, containing granular pigment, cellular *débris*, a few cells, and dark-colored urates.

It may be produced (1) by certain poisons, such as arseniuretted hydrogen, carbon monoxide, potassium chlorate, carbolic acid, and naphthol in large doses; (2) by acute infectious diseases, as, for instance, scarlet fever, typhoid fever, and malaria; and it is said also by (3) exposure to cold and severe exertion. (4) Hæmoglobinuria of the new-born is referred to elsewhere. (5) Paroxysmal hæmoglobinuria, a condition characterized by the occasional passage of urine containing free blood pigment, has been observed occasionally in children, generally in association with Raynaud's disease.

Pyuria.—Pus in the urine may be derived from the pelvis of the kidney, from the bladder, from an ulcer opening into the urinary passages, from the urethra in boys, and from the vagina and vulva in girls. Pus from the kidney is intimately mixed with the urine, which, except in pyelitis secondary to cystitis, is acid. Pus from

the bladder is usually accompanied by much ropy mucus, which is most copious towards the end of micturition. The urine is alkaline,[1] and passed at frequent intervals, the act being attended by much pain. Pus from the urethra passes before the urine; it can usually be squeezed from the meatus by manipulation, and micturition is accompanied by scalding pain. Pus from the vagina is mixed with flakes of lymph, and contains much vaginal epithelium, while the urine drawn off by a catheter, or passed immediately after thorough ablution, contains none.

Diseases of the Kidney.

During the pyrexial period of many **acute diseases**, diphtheria, typhoid fever, pneumonia, scarlet fever, small-pox, erysipelas, influenza, septic diseases, and acute enteritis, albumen appears in the urine in a large proportion of cases. It may be present on one or two days only and in very small quantity; but in other cases, especially in diphtheria and pneumonia, the amount may be large. As a rule, it disappears early; often, in fact, as soon as improvement in the general symptoms commences, and before convalescence is established. The urine may contain a little blood, but seldom in quantity sufficient to render it smoky. The course of the primary disease is not, as a rule, much influenced by the renal complication, though extensive granular degeneration of the epithelium will increase the risk of death from toxæmia.

The lesion of the kidney is a **diffuse nephritis**, which involves the elements of the cortex to different degrees in different cases. In the most typical form, that which is met with most often during the acute stage of pneumonia, scarlet fever, diphtheria, typhoid fever and small-pox, the kidneys are enlarged and full of blood, and there is marked *congestion* of the cortical substance. The capillaries of that part are dilated, and the glomeruli distended with blood, which may become effused also into their capsules and into the adjacent part of the uriniferous tubules. The cells of the convoluted tubules are in a condition of cloudy swelling, and the lumen contains leucocytes in a mucous matrix. In other cases, especially in erysipelas, septic fevers, and the early stage of scarlatinal nephritis, more rarely in diphtheria and typhoid fever, the inflammation is mainly *intertubular*. Leucocytes are found in large numbers between the tubules and around the glomeruli, the changes in the latter and in the cells being slight. The kidney is large, and pale or mottled. In the most severe cases the element which suffers most is the *renal epithelium*. The kidneys are large, smooth, and have a uniform yel-

[1] There is a peculiar form of cystitis, due apparently to the *bacillus coli communis*, in which the urine remains acid. It is usually a mild disorder, but is said to be attended sometimes by symptoms of typhoid type.

lowish or greyish surface on section. The cells are swollen, and granular from fatty degeneration. This "coagulation necrosis" is met with especially in the severest cases of diphtheria. It is due to the irritative action of toxic bodies, produced during the fever, or perhaps, in some cases, to the presence of bacteria. [The presence in the kidney of the Klebs-Loeffler bacillus, of the streptococcus, and of the staphylococcus pyogenes aureus, each alone and also associated with each other, repeatedly demonstrated at autopsy, makes it probable that the changes in this organ are due to a micro-organism. The streptococcus and staphylococcus pyogenes aureus have been found in the kidney in scarlet fever as well as diphtheria, occurring sometimes alone, sometimes together. Lemoine has found the streptococcus in the urine also of scarlatinal patients, 22 cases showing albuminuria, 1 showing no albumen.]

The diffuse congestive nephritis, described above as occurring during the pyrexial period and ceasing before convalescence or during its early stage, is sometimes succeeded by an acute or subacute **glomerulo-nephritis.** This occurs with particular frequency and severity after scarlet fever (post-scarlatinal nephritis), but may be observed also after typhoid fever, diphtheria, mumps, tonsillitis, measles, variola, varicella, pneumonia, acute rheumatism, osteo-myelitis, and other inflammatory affections, including, it is said, extensive simple impetigo. It may occur, also, as a complication of malaria. In some cases it follows, without any recognized antecedent illness, on exposure to cold, which has been assigned as the determining cause of post-scarlatinal nephritis. The kidneys are large and soft. In the early stage they are congested and full of blood, presenting often hæmorrhages at the surface and between the tubules. At a later stage they are pale, with a yellow or grey tinge, and present scattered white streaks between the tubules and prominent glistening granules, which are enlarged glomeruli. The enlargement of the glomeruli is due to a thickening of the capsule and proliferation of the cells, so that the cavity is distended by a mass of flattened cells which compress the capillary loops. The epithelial cells of the convoluted tubules are granular and detached in places, and the lumen of the tubules is filled with their *débris*, with leucocytes and with exudation. The epithelium of the straight tubules is less altered, but their lumen contains often opaque or hyaline cylinders. The arterioles, in relation with the glomeruli, are often involved in the inflammation, and may be obstructed. The lesions, therefore, are such as favor the occurrence of anasarca and uræmia—the transudation of water being hindered by the compression and contraction of the glomerular loops, while the excretion of toxic matters is diminished owing to the degeneration of the excreting epithelium.

Scarlatinal nephritis, which is the type of glomerulo-nephritis,

first, as a rule, produces symptoms which attract attention during the second week of the illness—that is to say, during the stage of desquamation. Not infrequently its appearance is delayed until the third, fourth, or even fifth week. In some cases the onset appears to be determined by exposure to cold. The symptom which is noticed earliest and is most characteristic is œdema, first usually of the eyelids in the morning, then of the conjunctivæ and face, then of the lower limbs, of the front of the abdomen and chest, and over the sacral region. Subcutaneous œdema may occur, indeed, in any part, and the variability in its degree and position is remarkable. It is soft, and long retains the impression of the finger. The skin is pale and dry. Fluid may be effused into the pleura, pericardium, and peritoneum. The meninges and brain are œdematous. Œdema of the glottis may occur suddenly and produce death. Albumen is, as a rule, present in the urine before anasarca develops, and continues after it has disappeared; but the anasarca may appear first, and it may even happen that at no time in the course of the case is albumen found in the urine. The quantity of urine diminishes progressively, and at the height of the attack only a few ounces, which may become almost solid on boiling, may be passed in the twenty-four hours. The urine usually contains blood, often in sufficient quantity to render it smoky. On microscopical examination, in addition to blood cells, hyaline and granular casts and epithelial *débris* will be found.

The *onset* of glomerulo-nephritis may be quite insidious, and anasarca makes its appearance without any premonitory symptoms. It may be preceded for a few days by headache, nausea, vomiting, and pain in the loins, and sometimes by slight pyrexia. **Eclampsia** may be an early, in fact, the earliest symptom, but more often the convulsions occur after anasarca has become marked. They are usually preceded and accompanied by great diminution in the quantity of urine, or by its entire suppression. The first attack is followed usually, at intervals of a few hours, by others. If, after the first, the child remains free for twenty-four hours, there is good ground for hope that no second attack may occur; but convulsions repeated at short intervals are of very bad omen, for, as a rule, fatal coma ensues. In some cases coma sets in without antecedent eclampsia. Death may be brought about also by serous effusion into the pleuræ, or by intercurrent pneumonia, pericarditis, or pleurisy. Dilatation of the heart is frequent, and death may occur from cardiac failure. Sudden loss or impairment of vision due to œdema of the discs or, more rarely, to neuro-retinitis, is an occasional complication of glomerulo-nephritis. Recovery of sight is as sudden, and is usually complete.

The *course* of glomerulo-nephritis varies very greatly. In some

cases it is a very mild disorder, lasting from four to six weeks, producing few symptoms beyond malaise, œdema, anæmia, and moderate albuminuria. In others it is a very acute and severe disease, bringing life into great danger in a few hours. In others, again, it runs a very chronic course, œdema coming and going, and albuminuria persisting in varying degree for many months. As a rule, if the patient escape uræmia, recovery takes place sooner or later; but, in the more chronic cases especially, the patient remains for long liable to fresh attacks on exposure to cold, or during any infectious disease from which he may suffer subsequently. In these more chronic cases the quantity of urine and the proportion of albumen which it contains, and the amount of urea eliminated, may vary very much from time to time, and even from day to day. During remissions the quantity of urine and of urea increases and the œdema diminishes, but the amount of albumen does not bear any relation to the other constituents of the urine. As a general rule, the larger the amount of albumen in the urine the greater the danger of the occurrence of uræmic complications. While the albuminuria continues the liability to a sudden exacerbation remains, and the longer the period during which albuminuria has persisted the less the expectation of ultimate escape from these dangers. In some cases granular contracted kidney is produced, more or less hypertrophy of the heart occurs, and the urine is copious and of low specific gravity. Albumen may be absent from the urine for long periods, or be present only in traces; but exacerbations, in which the urine decreases while the proportion of albumen it contains increases, are frequent.

[**Chronic Nephritis.**—It is now generally believed that while chronic interstitial nephritis is in childhood necessarily rare, chronic parenchymatous nephritis occurs more frequently than has been hitherto thought. It most commonly has its starting point in scarlet fever, though any of the infectious diseases may cause an acute nephritis which may run on more or less insidiously into the chronic form. A certain number of these cases are without apparent original cause, but careful and minute inquiry into the previous history will show in most instances the occurrence of some infectious disease, scarlet fever, diphtheria, measles, pertussis, influenza or tonsillitis, any one of which might have acted as an exciting cause of renal trouble. Chronic intestinal trouble also undoubtedly acts as a source of renal irritation, if not of actual nephritis.

The history is generally that since some acute illness, perhaps several years before, the general health has been impaired. It is unusual to hear complaints of any one specific symptom, but rather an account of general and indefinite malaise, capricious appetite, restless sleep, lack of energy, perhaps some slight œdema, pallor, headache, and often constipation. Physical examination may be abso-

lutely negative, except that usually we find a low percentage of hæmoglobin. It may be thought that the case is purely intestinal, and undoubtedly the digestive disturbance so frequently present aggravates the general condition, if indeed it have not been the primary source of all the trouble, including the nephritis. By some the renal condition would be regarded merely as a symptom, by too many it is overlooked, from failure to examine the urine. This should be a matter of routine in the examination of every patient seen, both in office and bedside practice, just as much as the examination of the throat, heart, or lungs. Nor should we dismiss the case with only one analysis of the urine. These cases are insidious, are apt to run an irregular course, and albumen and casts may be absent for periods of varying length of time, and unless we follow them carefully, the renal condition may escape us to smoulder on and to flare out with an acute exacerbation on exposure, *e. g.*, from invasion of the system by some infectious agent, such as the pneumococcus, streptococcus (causing a tonsillitis) or Klebs-Loeffler bacillus. However, if a chronic nephritis exist, repeated examinations of the urine will sooner or later reveal its presence by the detection of albumen, casts, and generally some fatty elements, such as fatty degenerated epithelium and occasionally fat globules, free or adherent to the casts. The casts are hyaline or fine granular, occasionally epithelial. The amount of urine varies, is sometimes normal, but reduced with an exacerbation of the disease; the specific gravity is generally lower.

The following two cases illustrate some of the points mentioned above and emphasize the importance of urinary analysis in the affections of childhood. The repetition of this point would seem trite, yet so much is it disregarded even to the point of criminal negligence that the reviser feels that it cannot be too strenuously insisted upon.

The first case is that of a boy 19 years old, first seen by me in July, 189–, and giving the following history: "delicate stomach" since 5 years of age, with flatulence, occasional epigastric distress and tendency to constipation, "hip trouble" at 5 years, measles at 7 years, "la grippe" at 18 years, with marked tonsillar symptoms. The urine had never been examined.

Now for three days, general abdominal pain and tenderness in the right iliac region, some nausea, little vomiting, constipation, elevation of temperature.

Physical examination showed a young man well developed and nourished, face flushed, tongue thick white coat, throat negative. *Chest*, negative. *Abdomen*, general tenderness, most marked in the right iliac region, no dulness, no mass felt. No œdema.

T. 102°, P. 84 not otherwise abnormal, R. 20.

Urine.—High, acid, 1,026, albumen 1 per cent. (single specimen). Sed. slight: little free fat, few blood-corpuscles (normal), occasional

renal cell; few casts; hyaline with fat globules and epithelium adherent, 1 or 2 epithelial; some of the casts long, others short and of large diameter.

Diagnosis.—(*a*) Appendicitis, though symptoms may be due to an acute exacerbation of a (*b*) Chronic Parenchymatous Nephritis.

The subsequent course of the disease showed it to be an appendicitis which went on to recovery without operation. The symptoms at first all pointed to appendicular trouble and the urine was examined merely as a routine habit and revealed the above condition. It had never been examined before, either at the time of the attack with measles, with hip-trouble, or with "la grippe" one and a half years previous.

Speculation as to the origin of the nephritis is of course idle. These possible causes may have produced it: the attack of measles 12 years previous, the attack of "la grippe" $1\frac{1}{2}$ years previous, or the intestinal trouble chronic for 14 years. The last named, if not the actual cause, undoubtedly aggravated the renal condition.

The case continued in my care for about a year, at times the albumen decreasing to a mere trace, the casts and fatty elements entirely disappearing, all abnormal elements, however, returning on the many indiscretions indulged in. At the end of that time, the advice of a "christian" so-called "scientist" was sought, who failed however to banish the albumen, casts and fatty elements from the urine, and in a few weeks he returned to a physician who reports that his present condition 3 years later is good, but that albumen and casts are still occasionally found in the urine.

The second case is that of a boy 7 years old, who, coming from out of town, presented himself at Dr. Cotton's clinic at the Rush Medical School in April, 189–. He had had diphtheria the preceding December followed by extensive post-diphtheritic paralysis involving all the limbs, for which he had received energetic tonic treatment, with a diet rich in proteids. Electricity had also been administered, but little improvement had been made. In March he had an attack of "la grippe," a month after which he was brought to the clinic. He then was poorly nourished, walked and used upper limbs with much difficulty; no œdema. Heart and lungs negative. The urine showed albumen $\frac{1}{2}$ per cent., fatty renal epithelium, free fat and casts, fatty, hyaline and fine granular. The urine had never been examined before. The case reported but once at the clinic and has never been seen since.

These two cases illustrate how much we may learn from ordinary analysis and to what disastrous results unrecognized renal trouble may lead.]

Treatment.—The patient should be kept in bed and given a fluid diet, consisting of milk and whey, with barley water, lemon water,

and Imperial drink as beverages.[1] Beef-tea and other broths which contain toxic extractives should be excluded. At the beginning of the illness a drastic purgative should be given and repeated in two days, a mustard plaster or the dry cup applied over the loins, and a hot bath given to promote diaphoresis. Subsequently the wet or dry pack administered daily is useful, or in more severe cases the hot air bath. Caffeine is to be preferred to digitalis if there be signs of cardiac failure, and is useful also as a diuretic. The acetate of ammonia and other alkaline diuretics, and the benzoate of soda are also of value, but hypodermic injections of pilocarpine should be used with great caution, if at all, in children. When improvement commences, the patient should still be kept in bed for several weeks and given a fluid diet, of which milk—which has a diuretic action—should form a large part. Preparations of iron, either the perchloride or the acetate, or the citrate of iron and ammonia, should be given in full doses. The routine use of purgatives with the object of diminishing dropsy is not advisable. Even after the œdema and albuminuria have quite disappeared the patient should be regarded as an invalid for some months, and should not be allowed to run the risks of school life, especially at a public school. Cold and damp should be guarded against, and if possible the winter season should be spent in a warm climate. Later, a residence at a high altitude, especially if it can be combined with the drinking of a chalybeate water, is advantageous.

Amyloid degeneration of the kidney is generally a part of widespread amyloid disease secondary to some condition such as bone disease, which is liable to cause prolonged suppuration, but it may occur also in the later stages of chronic parenchymatous nephritis.

The organs are pale, smooth, firm on section, and usually enlarged. The cortex is pale and glistening. The glomeruli in which the amyloid change begins are prominent; the pyramids are of a dark red color.

The urine is increased in quantity, of low specific gravity, pale in color, and contains usually some albumen, often only a trace, and hyaline or finely granular casts. Dropsy occurs in most cases. It affects usually the lower extremities, but may be extensive. The patient becomes very anæmic and cachectic, suffers from diarrhœa, and succumbs usually to asthenia, or to an intercurrent malady.

The **diagnosis** must rest on the occurrence of albuminuria and polyuria in the course of a disease liable to produce amyloid degen-

[1] [*Imperial Drink.*

Cream of Tartar,	℥ss
1 lemon cut in slices,	
White Sugar,	½ lb
Water,	Oiij

Mix together and let them stand for half an hour. Appendix.]

eration. Enlargement of liver and spleen will confirm the diagnosis.

The **prognosis** is very bad, since amyloid degeneration occurs usually in patients whose strength has already been reduced by long illness.

The **treatment** must be directed, primarily, to the removal of the cause and, secondarily, to guarding the patient against chill and against accumulation in the blood of toxic products derived from the food.

Congenital cystic disease of the kidneys, a rare condition, is due to persistence and abnormal development of portions of the Wolffian body. It should be noted, however, that cystic disease of the liver may also be present. The cysts vary in size and number, and may have produced so great an enlargement at birth as to obstruct delivery. In such cases the infant is still-born, or survives but a short time. On the other hand, the enlargement of the kidney may only be discovered in middle age. The organs consist of a collection of cysts of varying size, and, to the naked eye, may show no appearance of kidney structure, though this will be discoverable on microscopical examination. The symptoms ultimately produced are those of chronic fibrosis.

Uric acid diathesis.—It is very common for infants and children under the age of two or three years to pass water which quickly becomes turbid from separation of urates, or which is even turbid at the time of passing. Such precipitation may be associated with a diminished bulk of urine, due to the ingestion of too small a quantity of fluid, or to an unusual loss of fluid, either by the skin or the intestines. Large quantities may appear also during convalescence from acute diseases, especially scarlet fever. Such occurrences are more or less of an accidental nature, and ought not to be regarded as evidences of the existence of a uric acid diathesis. On the other hand, the repetition at frequent intervals of these accidents points to defects either in diet or in metabolism. There can be no doubt that children of gouty parentage are particularly liable to such attacks, and that in addition they on some occasions pass uric acid crystals. Whereas the passage of urates may give rise to no symptoms, it is otherwise with uric acid.

Sir William Roberts enumerates, as the conditions favoring the separation of uric acid from the urine, high acidity, a small amount of pigmentation, poverty in salines, and a high percentage of uric acid. In infants the quantity of pigment is small, and the percentage of uric acid is said to be high as a rule, or as a frequent incident. When, therefore, there is added to these conditions an undue acidity of the urine, three of the four main factors of the precipitation of uric acid in the urinary passages will concur. In older children, especially among the poor, who live largely upon cereals, the fourth

factor—poverty in salines—may also be present. The separation of the uric acid may occur at any part of the urinary tract, in the terminal straight tubules of the kidney, in the pelvis, the ureter, or the bladder. In a typical attack, the child rather suddenly becomes restless and peevish. It screams when touched, and it may be possible to detect some tenderness in both lumbar regions. Micturition is frequent and attended by pain. The urine deposits uric acid, which may be recognized as a cayenne pepper deposit in the vessel, or as red stain on the napkins. The external orifice of the urethra is red and excoriated, and in girls there may be much irritation of the vulva. In boys with phimosis, balanitis may be produced with much œdema of the surrounding tissue. The attack subsides in a few days, but similar attacks are very apt to occur until the child reaches two or three years of age. For the next two or three years of life the liability to such attacks appears to be less, but after five or six years old they again become frequent, and the irritation produced by the presence of uric acid in the urine is one of the causes of enuresis.

The etiology of the condition is somewhat obscure. The attacks may be determined by the ingestion of too liberal a diet, by want of exercise and fresh air, especially during winter months, when children are much confined to the house, and they are favored by too small a quantity of fluid in the diet. Such attacks occur not only in children who inherit the gouty diathesis but also in those of strumous type.

Calculus in the pelvis of the kidney is not very rare in childhood. It usually consists of uric acid, more rarely of oxalate of lime. It may produce few or no symptoms, though there may be blood in the urine, which in older children may give to it a well-marked deep red color. In infants a stain of bright blood is noted on the napkin. Renal calculus is said to be the commonest cause of hæmaturia in infants. In other cases the irritation of the stone may eventually induce suppurative pyelitis. Infants may suffer from true **renal colic.**

Gibbons, who has given an excellent description of this condition,[1] has met with it only in private practice and in the children of gouty parents. The symptoms are pronounced, though the diagnosis is often difficult. In the midst of apparent health, it may be during sleep, the child is seized suddenly with acute abdominal pain, accompanied perhaps by vomiting. The temperature may be normal or raised 2° or 3° F. It resents any disturbance or examination, lies persistently on one side, and it may be possible to ascertain that the loin on the opposite side is acutely tender. After a time the child may become prostrate, or even collapsed. The attack subsides in from one to two days, and convalescence is rapid. Similar attacks,

[1] *Medico-Chirurgical Trans.*, vol. lxxix., p. 41.

to the number of three or four, may occur at intervals of a few weeks or months. During the attack the legs are drawn up and the thighs flexed upon the abdomen. One or both testicles are retracted. (Five out of six of the cases recorded by Gibbons occurred in boys.) The urine passed during the attack is clear, but contains a trace of albumen, and, under the microscope, free blood cells and crystals of uric acid. After the attack the urine may contain large quantities of uric acid, either in detached crystals, or in small masses aggregated together by mucus.

If a calculus reach the bladder it may be passed by the urethra. This, as might be expected, occurs more often in girls than in boys. In boys, the calculus may become impacted in the urethra. If retained in the bladder, it gives rise to frequent painful micturition, arrest of the stream, passage of blood at the end of the act, pulling of the prepuce, and other ordinary symptoms of vesical calculus.

The **treatment** of excessive excretion of uric acid must depend upon a recognition of the cause and the nature of the symptoms. The frequent passage of uric acid in the free crystalline state should direct attention to the diet and the condition of the digestive organs. It may be possible, by diminishing the amount of proteids, if this be excessive, to remove the cause, or it may be found that the child is suffering from acid dyspepsia, due, perhaps, to fermentation of an excessive quantity of carbohydrates. The administration of alkaline medicines, which will be desirable during the attacks, should not be continued in the hope of warding them off. In infants, a deficient amount of fluid in the diet is not likely to be a cause of the deposition of uric acid, but in older children an increase of the fluid taken may serve to prevent attacks. Renal colic should be treated by giving a warm bath, followed by a poultice to the loins, and the administration of a mixture containing compound tincture of camphor, ammonium bromide, and lithium carbonate. If any doubt exists as to the possibility of the presence of vesical calculus, the bladder should be sounded carefully, and, if the symptoms persist, the examination should be repeated.

The prophylaxis of the consequences of the uric acid diathesis is important. The child, if an infant, should be well clothed, taken out into the open air daily, and the diet regulated to avoid dyspepsia. To older children the same remarks apply, with the addition that they are benefited by taking as much exercise as possible, short of over-fatigue.

Hydronephrosis.—Dilatation of the pelvis of the kidney caused by accumulation of fluid may be *congenital* and due to defect in development of the ureter or urethra. The dilatation at birth may be so great as to produce a distension of the abdomen, which obstructs delivery. In another form the dilatation affects the pelvis and

ureters, but there is no obvious obstruction. In such cases the dilatation is moderate, and the patient may survive for some years, but eventually succumbs, usually to purulent infection of the dilated parts.

Intermittent hydronephrosis may be met with in children. Uneasiness or an attack of pain in one or other flank is found to be associated with the development of a tumour in the kidney region, which may attain a large size and then suddenly disappear, its disappearance being attended by cessation of the pain and the passage of a large quantity of pale urine.

Acquired hydronephrosis is not common in children. When of moderate size, it is usually possible to recognize that the tumour formed by hydronephrosis is renal, but it may be difficult to say whether it be due to sarcoma or fluid. When somewhat larger, so that it reaches to the middle line, the question of retro-peritoneal sarcoma will arise, and the question may only be possible of solution by puncture, which, in hydronephrosis, yields a clear, faintly yellow fluid, containing urea and uric acid in small quantities. When it is considered necessary to make a puncture, the needle should be inserted over the tumour posteriorly, midway between the iliac crest and the twelfth rib. The best treatment appears to be by incision in the lumbar region and drainage. The treatment of intermittent hydronephrosis is not settled. When associated with movable kidney, as is sometimes the case, a properly adjusted pad to keep the organ in place may give much relief. Movable kidney, however, is very uncommon in childhood.

Pyelitis is in children due usually either to stone or to tubercle, but it occurs occasionally in the course of specific fevers, and is met with, in rare instances, without any discoverable cause. It is also occasionally secondary to perinephritis. The symptoms, if the affection occur in the course of a specific fever, are not very distinctive, and, unless the urine be examined, will probably be overlooked. The urine may be smoky from the presence of blood. The amount of pus present varies, and it may even disappear for a time owing, probably, to temporary blocking of the ureter. In uncomplicated cases there is intermittent fever, the exacerbations being accompanied by rigors, so that the case may resemble malarial fever. The child looks ill and, if the condition persists for some weeks, becomes much emaciated and exhausted. There is tenderness in one flank, and enlargement of the kidney may be demonstrable; eventually great distension of the renal pelvis may occur. In the treatment of this affection, astringents appear to be of no use, and the best course is to give the patient large quantities of water containing a little citrate of potash, or some of the milder alkaline mineral waters to drink. When the enlargement is sufficiently great to

allow a distinct tumour to be made out, or, if the general symptoms are persistent and severe, the kidney should be explored.

Perinephritic abscess.—Inflammation and suppuration around the kidney may be the result of injury, or of extension of inflammation from the pelvis or ureters, or from the appendix, spine, or pleura. In other cases, the condition occurs as a complication or as a sequel of acute infectious diseases; but in a considerable number of cases no cause can be discovered. The inflammation produces a large, ill-defined swelling in the flank; the swelling is very tender, and there may be much pain, which is greatly aggravated by movement. The patient keeps the thigh flexed, and there may be a considerable resemblance to early hip-joint disease. It will be found however that with the hip flexed gentle passive movement of the joint does not cause pain. The formation of pus is attended and followed by irregular fever, rigors, and sweats. The only treatment which can relieve the patient is incision and drainage; the operation should not be too long delayed as the pus may track downwards into the groin, or the abscess may rupture into the peritoneum, bowel, bladder, vagina, or even into a bronchus.

Tumours of the kidney, though rare at any age are more common under six years of age than subsequently, and are probably the form of new growth most often met with in the abdomen in children under that age. Their pathology is obscure, but they are probably in all cases congenital. Adenomata may be met with, but as a rule the growths are sarcomata or rhabdo-myomata, that is to say, round-celled sarcomata containing striated muscular fibres. In most cases, the first symptom to attract attention is enlargement of the abdomen, which is found to be due to a tumour in the lumbar region. It is at first, as a rule, freely movable on palpation, but is little affected by respiration. It has a smooth, rounded or indistinctly lobulated surface, and when soft and of very rapid growth may fluctuate. Hæmaturia is present in about one-third of the cases and may be the earliest symptom. The urine contains clots of blood which sometimes have the form of casts of the ureter or pelvis; their passage produces attacks of colic. The growth of the tumour, which may attain an enormous size, is attended by rapid emaciation. An important point in the diagnosis of all renal tumours is the fact that they are traversed by the colon, which usually yields a tympanitic note; when, however, the enlargement is great, the gut may be so much compressed that this sign is wanting. If the case be seen early, renal may be distinguished from retro-peritoneal sarcoma by the fact that the latter at first forms a tumour in the middle line and is not movable; but at a later stage it may be impossible in the absence of hæmaturia to make the distinction. An enlarged spleen is more movable than a renal tumour, moves freely with respi-

ration, and presents usually a sharp edge and notch. On the right side, tumour of the kidney may present some resemblance to tumour of the liver, but may be distinguished by the slighter movement with respiration, and, as a rule, by a band of resonance between it and the lower edge of the ribs. Abscess or tuberculous disease of the kidney usually causes pus in the urine and pain and tenderness in the lumbar region.

Tuberculosis of the kidney, except as a part of a general tuberculosis, is rare in childhood, but cases occur occasionally in which tuberculous pyelitis of chronic type is met with, and leads to disorganization of the kidney. The course in such cases is very insidious, and pain may only be complained of when the pelvis has become distended with purulent and cheesy matter; an enlargement of the organ may then be detected, and, as a rule, the urine contains albumen and pus. If the ureter becomes blocked, pyonephrosis ensues, and a fluctuating tumour forms. In such cases, the temperature is hectic, and the symptoms can only be relieved by operation. The occurrence of symptoms of pyelitis accompanied by hemorrhage points rather to stone in the kidney. Short of operation, which is rarely called for in children, treatment should be directed to rendering the urine as unirritating as possible, and for this purpose a milk diet is the most efficacious.

CHAPTER XXXVII.

DISEASES OF THE NERVOUS SYSTEM.

The Nervous System in Infancy—Night Terrors—Headache—Stammering and Stuttering—Alalia—Deaf-Mutism; Forms; Causes; Prophylaxis; Treatment.

The nervous system at birth has not attained its full structural development, and its functions are imperfectly developed. The brain is large at birth in relation to the size of the body generally. After birth it grows in bulk with great rapidity, and the convolutions become more complex and the sulci deeper. This period of very rapid growth terminates about the seventh year, and thereafter the increase in bulk is much slower. The spinal cord is slender, and the pyramidal tract very imperfectly developed at birth.

The special characteristic of the movements performed during the earliest infancy is the want of coördination. The main exception is the act of sucking, which is perfectly performed within the first three days of life, and is therefore regarded as an instinctive movement. The act of grasping is also apparently instinctive. The reflex movements gradually become more numerous and complicated; thus sneezing and coughing may be performed at birth, but tickling does not produce smiling until the end of the second month. The skin reflexes are present at birth, and the tendon reflexes (knee-jerk) also, as a rule. The infant also performs certain spontaneous movements—crowing, crying, kicking, and waving its arms from an early age. They are apparently elicited by general "large" somatic sensations of comfort or discomfort. The power to coördinate voluntary purposive movements is acquired slowly. Thus grasping is first performed as a voluntary act about the fourth month, at which age both instinctive and voluntary grasping may usually be elicited in succession in the same child. Voluntary grasping movements are at first very irregular and imperfect. The infant is also handicapped in its effort to grasp an object by inexperience in judging distance. Accommodation is imperfect, and the coördination of the ocular muscles is not complete, so that irregular movements often occur during the first few weeks of life and produce transient squint. This squinting is particularly apt to occur on awaking from sleep. During the first two years of life the child is undergoing an extremely rapid process of education. It learns to distinguish objects

by taste, sight, and touch ; it learns to distinguish between animate and inanimate objects, and to know certain individuals. It learns to judge distances, to grasp and pull with appropriate force, to walk, and to speak. For the next two years education is only a little less rapid. The brain of the infant and child then are in a continuous state of active development and, during waking hours, of intense functional activity. Owing to the imperfect development of the inhibitory and regulating apparatus, the response is out of proportion to the strength of peripheral stimuli, and the area of response is apt to be unduly wide.

In the sphere of the emotions there is a similar want of control. The intellectual processes are slow, and the power of discriminating between the objective and subjective imperfect. This is well exemplified in the **night terrors** (pavor nocturnus), so common in childhood, especially in neurotic children, or those habitually subject to over-excitement. The condition is closely analogous, if not identical, with nightmare, and of those vague feelings of fear which make many adults dread to pass a churchyard by night—that instinctive and therefore unreasoning dread of darkness doubtless inherited from remote ancestors, which makes "two o'clock in the morning courage" the highest form of that ancient virtue. Most children dislike the dark, and dread being set to go to sleep in a dark room. In children subject to night terrors this feeling is greatly exaggerated. The attack of pavor nocturnus generally begins from one to three hours after falling asleep.[1] The child wakes up suddenly with a shriek or loud cry, and appears much alarmed. It seems to have visual, more rarely auditory hallucinations, and, though not completely unconscious of its surroundings, it does not recognise the persons about it. The heart is found to be acting violently, the pulse is rapid, the limbs tremulous, and the body is covered with sweat. In a few minutes, or perhaps only after half an hour, it begins to grow calm, loses its hallucinations, recognises those about it, and soon falls into a sleep, which usually lasts undisturbed until morning. Occasionally such attacks occur by day if the child fall asleep. Children who suffer in this way are always of neurotic type, usually have a neurotic parentage, and often have dyspepsia or chronic diarrhœa. The liability to pavor nocturnus begins in the second year, and is rather greater in the male than in the female sex. The attacks gradually become less frequent and generally cease altogether about twelve years of age. In a few cases the patients have become epileptic, but this was probably no more than a coincidence. Children liable to these attacks should be treated with the greatest consideration. They should not be compelled to go to bed in the dark, and they should not be frightened by silly stories of the supernatural, in which some

[1] Braun, *Jahrb. f. Kinderhkde.*, Bd. xliii., S. 406.

nurses delight. They should get enough sleep; very often it will be found that they are allowed to sit up late. They should be carefully dieted, and in particular any imperfection in the digestive processes should be corrected. In some cases there is marked constipation or irregular action of the bowels, with occasional periods during which the stools are fluid and very offensive. In such cases attention to the diet and the use of antiseptic drugs internally (see p. 387) will prevent the recurrence of the night terrors. Sedative drugs seldom have much influence in preventing the terrors, and opium is distinctly contra-indicated.

Headache is not a common symptom in infants or young children, and when its presence can be established it will be found, in many cases, to be due to disease of the ear, cranium, meninges, or brain.

Children may suffer from *toxic* headache due to ill-ventilated rooms—one variety of morning headache is due to this cause—or to absorption from decomposing material in the intestinal canal. There is also that large class of toxic headaches, which are to be observed frequently at the onset of scarlet fever, measles, typhoid fever, and other acute diseases. Mistakes, however, are not likely to occur in such cases. It is otherwise with the headache produced by *error of refraction*. Of these the most important is hypermetropia; it comes on in the morning as soon as the eyes are opened, but wears off if the eyes are not used for near work. Reading or sewing makes it worse. It appears to be due [1] to spasm of accommodation. During sleep the ciliary muscle is at rest, but upon opening the eyes in the morning it is thrown into strong action, and the suddenness of the transition causes pain. When, after a time, adaptation takes place the pain diminishes, only to increase again if a fresh call is made on accommodation by near work. The pain, which is accompanied by superficial tenderness, is referred to the mid-orbital area, which is situated over the centre of the eyebrow and includes a greater part of the upper lid. The longer the strain the larger the part of the area involved, and when most developed the pain may be referred also to an area higher up, at a point about the margin of the hairy scalp in a vertical line above the eyebrow area. The patient states that the pain is "over the eyes" or "in the eyes," and, to point it out, places the right hand across the forehead, touching the centre of the right eyebrow with the hypothenar eminence, and the centre of the left eyebrow with the tips of the fingers. For this reason the headache has been spoken of as "frontal," but if an intelligent patient is told to point out the painful areas with both hands, he places the tips of the fingers just above each eyebrow.

The combination of astigmatism with hypermetropia increases greatly the liability to headache. A robust child may have a good

[1] Head, *Brain*, vol. xvii., p. 339.

deal of hypermetropia (+ 3D) without headache, whereas an astigmatic error of + 1D may produce definite pain and tenderness. Deterioration of general health, and "tone" may, even in simple hypermetropia, be followed by headache. Myopics, after long use of the eyes, may complain of an ill-defined, tired, aching feeling in the forehead; but myopia, especially in children, is not of any importance as a source of headache, except in the rare cases in which it is complicated by spasm of accommodation. Headache in this situation, which is worse in the morning and aggravated by close work, especially in a child who has recently been set to much reading, writing, and sewing at school, should raise the suspicion of hypermetropia. Paralysis of accommodation by atropine will remove the headache, and will also serve as a necessary preliminary to the estimation of the error of refraction.

A common cause of pain referred to the side of the head and neck is otitis media, but this may also cause pain towards the vertex, and in the parietal regions if there be a rise of tension in the middle ear such as takes place before the drum is perforated. A common cause of dull, heavy pain in the frontal region is the presence of adenoids in the naso-pharynx. Headaches from anæmia are not very common in children, but in girls of the rheumatic diathesis they occur rather frequently, and are sometimes the precursors of chorea. True megrim is a rare affection in childhood, but headaches associated with the presence of the uric acid diathesis are common, and unless their real nature be recognized are apt to be intractable. Many patients who would be cured by a dose of calomel, or a short course of magnesium sulphate, go on for years taking bromides, phenacetin, and other hypnotics with only temporary relief.

Stammering and **stuttering** are due to a want of regular contraction and coördinated action among the muscles concerned in articulation. This imperfection may exist only in the lips, interfering with the production of the explosive consonants (*b*, *p*, *d*, *t*, hard *g*, and *k*) or the tongue may be involved so that there is hesitation in the production of the continuous consonants (*v*, *f*, *th*, *z*, *s*, *sh*, *y*, *w*, *m*, *n*), or again there may be laryngeal spasm causing difficulty in the production of the vowel sounds. To these conditions some would limit the term *stammering*, and would apply the term *stuttering* only to those more distressing cases in which, in addition to the affection of the neuro-muscular mechanism of articulation proper, the respiratory muscles are involved.

Defects of speech of this type are often hereditary, or they occur in families in which one or other parent is distinctly neurotic. As a rule the defect does not become marked until after the age of three or four years. Sometimes it is perceived first after a severe febrile illness (infectious fevers), occasionally it is associated with chronic

naso-pharyngeal affections and is improved after the local condition has been treated. A mild degree of stammering may be produced by imitation, and cases are sometimes met with which are hysterical, or in which the hysterical element counts for much.

Difficulty with the explosive consonants is the commonest form, but both this and other forms only become serious affections when combined with irregular action of the muscles of respiration. In such cases, during the attempt to breathe, spasm of muscles of the face, arms, and trunk may occur and increase greatly the distress which the patient suffers.

The essential point in **treatment** is to give the child regular and systematic instruction in breathing and articulation, by exercises repeated many times a day. Considerable skill and much patience are demanded, and it is best, if possible, to commit the child to a teacher with special experience and aptitude for such work. The difficulty in speech is usually absent during singing or intoning, and in slight cases ordinary singing lessons, if the teacher give attention to the breathing, will often suffice to produce great improvement. On the other hand, the individual may learn to sing and intone well, although no improvement has taken place in the speaking utterance.

Cases are occasionally met with in which the child, though apparently well developed and intelligent, does not acquire the art of speech. This condition of **alalia idiopathica** may be due to structural defect of development, and be permanent. It is then associated, as a rule, with other evidences of idiocy. But it is not uncommon to meet with children who, up to the age of three or four years, make little or no effort to articulate. This retarded development is a source of great anxiety to the parents. The diagnosis may generally be made without much difficulty by observing the general aspect and habits of the child, who learns to play and to walk like other children. Such children when they begin to utter articulate sounds learn to speak with great rapidity. A child who reaches the age of two or three years without attempting to speak should receive regular instruction for short periods several times a day.

Aphasia due to organic disease may occur in childhood and presents the same characters as in the adult. The prognosis is, however, better (see p. 87).

Deafness may exist in any degree from mere "hardness" or dullness of hearing to total loss of the sense. Any degree of deafness great enough to prevent the human voice, used in the ordinary manner, from being heard will, if it be congenital, or developed in childhood, render the child a deaf-mute, unless this result be prevented by special education. The term *surdism* is applied to those degrees of deafness which make "the acquisition of speech in the very young impossible by ordinary means, or which involve the loss

of recently acquired speech."[1] Deaf-mutism is rather more common in many boys than girls.

Few deaf-mutes are totally deaf. A large vibrating tuning-fork in contact with the cranium is heard by almost all. Aërial sounds —for instance, a tuning-fork at a short distance—are heard by all but about 10 per cent. Hearing for speech to an extent sufficient to be of use in teaching exists in about 25 per cent. of deaf-mutes.

About half the deaf-mutes are deaf from birth. Deaf-mutism may undoubtedly be hereditary. Thus to a deaf-mute parent (father or mother) may be born some hearing and some deaf children, or a deaf-mute child may be born to hearing parents if one belong to a family in which congenital deafness has occurred in previous generations, even though not in the direct line. In other families deaf-mutism is one term of a series of nervous defects, such as idiocy, insanity, or epilepsy, with which various members are afflicted. Consanguinity increases the liability to deaf-mutism in the offspring,[2] and is more potent in this respect than the marriage of deaf-mutes, since in one parent the deafness may have been acquired. Unions between deaf-mutes are commonly not prolific, and are often sterile.

Congenital deafness may be due to *imperfect development* of the organ of hearing. Thus malformation of the auricle may be associated with almost entire absence of the structures of the internal ear. But either the middle or the internal ear may be affected independently of the other. In other cases, probably the majority, the deafness is due to *inflammation* of the internal ear either before birth or shortly after, ending in bony overgrowth and destruction of the nervous mechanism.

Acquired deaf-mutism is due, in the majority of cases, to inflammatory disease which has spread from the middle to the internal ear, and caused destruction to the membranous labyrinth, and the nervous structures in relation with it. In other cases the inflammatory mischief has extended from the cranial cavity, and in a few the lesion producing deafness is a primary inflammation of the labyrinth. In any case if the disease which causes deafness occurs before the age of six or seven the child is likely to become mute. The liability will be increased if the disease which causes the deafness has produced also other changes which have lowered the general intelligence; it will be diminished if the child has already possessed fair powers of speech, and if well-directed efforts are made to train the child to retain and extend the powers it previously possessed.

[1] "Deaf-Mutism," by J. Kerr Love, M.D., and W. H. Addison, A.C.P., Glasgow, 1896. This is an excellent study of the subject.

[2] According to statistics said to have been collected by Liebreich, there were in Nassau among the Roman Catholics, who forbid consanguineous marriages, one deaf-mute to 1,397 persons living, while among the Jews, who encourage such marriages, there was one deaf-mute to 508.

In Great Britain deaf-mutism dates from an attack of scarlet fever, measles, typhoid fever, or whooping-cough in 44 per cent. of all cases of acquired deafness; scarlet fever is alone responsible for 23.5 per cent. Next in importance to the infectious fevers stands meningitis and various diseases of the brain (23.9 per cent.). Falls and other accidents are held responsible for nearly 9 per cent. In America and on the Continent of Europe the ratios are different, owing in part to the fact that an unusually large portion of children attacked by epidemic cerebro-spinal meningitis, a disease very rare in Great Britain, become deaf.

Prophylaxis.—The prevention of the more serious consequences of otitis occurring as a complication of the acute exanthemata is therefore of great importance in this connection. The necessity for the early systematic treatment of otitis from other causes is also great, since a case which may be amendable to treatment in the early stage may be most intractable after the chronic inflammation has persisted for three or four years.

Treatment directed to the naso-pharynx, the removal of adenoid vegetations when they interfere with the ventilation of the middle ear through the Eustachian tube, and of enlarged tonsils is also required in many cases, for every effort should be made to improve such power of hearing as the child may have. Search should be made for any obvious cause of deafness, otorrhœa, adenoids, etc., in order that the condition may be treated in the hope that hearing may be so far improved that the child may be, in part at least, instructed through this sense. The education of deaf-mutes is now compulsory both in England and Scotland. There is much difference of opinion as to the best method of education. For those children who possess a sufficient remnant of hearing to be instructed through the human voice there can be little doubt that this method should be employed as much as possible. For those who have no useful hearing the so-called oral system is the best, if they possess sufficient intelligence. Under it the pupil learns to understand speech by watching the movements of the lips, and acquires the power of speaking with more or less distinctness. With those who possess some hearing power the two systems—the acoustic and the oral—may be combined. There can be little doubt that many deaf-mutes, whether the defect be congenital or acquired, are of a low order of intelligence. Many are of average intelligence, and a few possess very superior abilities. When the child does not possess sufficient quickness to acquire the art of lip-reading, it may yet learn to understand others, and to express itself by means of the well-known hand signs, and the power of using this system is a useful possession for all deaf-mutes. The education of a deaf-mute should begin at about seven years of age, and the oral system requires a

course of instruction extending over ten years. The teachers must undergo a special training,[1] and be endowed with much patience. The more individual attention the teacher can give the better the result.

[1] There is a training college for teachers at Ealing, Middlesex, and also at Fitzroy Square, London. There are 31 public schools for deaf-mutes in England and Wales, with (in 1895, according to Love and Addison) 2,630 pupils, 10 in Scotland with 524 pupils, and 4 in Ireland with 547 pupils. According to the returns of the census of 1891 there were nearly 20,000 deaf-mutes in the United Kingdom at that time. If the children under five, in whom the defect has not yet been recognized are added, it is estimated that the number would be about 22,000. The proportion to the general population appears to be decreasing slowly.

CHAPTER XXXVIII.

HYSTERIA: PICA.

Hysteria: Definition; Somnambulism; Delirium; Paralysis; Neuro-mimesis; Fasting Girls—Diagnosis of Hysteria—Treatment—Pica or Dirt Eating.

Hysteria is a psycho-neurosis. It is the manifestation of a special form of degeneration traceable to the influence of heredity. As Donkin has well said,[1] "it must be remembered . . . that some degree of mental disorder, evinced in the sphere of feeling rather than of intellect, colors and underlies all its phenomena, predominantly psychical in expression though they often are."

Somnambulism, which is relatively common in childhood, especially in girls, is, I believe, usually, if not always, an hysterical phenomenon. This opinion, advanced by Gilles de la Tourette,[2] and accepted by Charcot, is supported by, among other considerations, the facts that the somnambulistic state is often preceded by slight convulsions, and that somnambulistic children at a later age in many cases show distinct signs of the hysterical constitution. The patient while in the somnambulistic state performs purposive acts as well as when awake, or with even greater sureness and dexterity, but on returning to the normal state has no memory of these acts. The functions of the higher centres concerned in consciousness are suspended, but those of the lower automatic centres are in full activity. Somnambulism begins usually during sleep, whence its name. The similarity between somnambulism and the post-epileptic automatic state will not escape attention. The parallelism between epilepsy and certain manifestations of hysteria is indeed so remarkable that it often creates great difficulty in diagnosis.

A common manifestation of hysteria in childhood is **delirium** preceded or not by distinct convulsions, and generally accompanied by struggling, biting, kicking, crying, shouting, etc. In some cases there is a well-marked tonic stage, and opisthotonos may be produced. There does not appear to be complete suspension of consciousness, but the assertion of the patients that they have not any accurate

[1] "Diseases of Childhood (Medical)." London: 1893, p. 310. Those who would pursue this subject further cannot find a more trustworthy guide through the labyrinth than the article by the same author in Tuke's "Dictionary of Psychological Medicine."

[2] "Traité de l'Hystérie," sec. part., t. ii., p. 300.

recollection of the incidents of their attack is probably true. The delirious state may last for a few minutes, for hours, or days, and when long lasting is commonly succeeded by a stage of depression. At the moment of onset there is a sudden pallor, but later the face is flushed, often perspiring; afterwards it wears a dull, heavy expression, and there is usually some congestion or actual cyanosis. During the delirious state choreiform movements may be conspicuous, and may persist for some days. The subjects of attacks of this nature, usually girls of eight or nine years old and upwards, are often bright and intelligent, but excitable and emotional, and some emotion such as a disappointment or an injustice, real or fancied, at school, is commonly an immediate antecedent.

Paralysis is rare in childhood, and commonly there is only partial loss of power in the parts affected. Hemiplegia, monoplegia, and paraplegia have been observed. The onset may be sudden after convulsions, but is usually gradual. The duration is very uncertain, and the palsy may disappear suddenly or gradually under the influence of some new emotion, or of some fresh circumstance which calls the will into exercise. Paralysis, if persistent, is usually accompanied by **contracture**, and when this has lasted for some time the tendon reflexes become exaggerated and there may even be ankle clonus, unless the rigidity be very great. Contractures of the muscles of the leg may produce various forms of talipes, usually symmetrical. Hysterical aphonia may occur in young girls, the patient speaking in a whisper, or the child may refuse to attempt to speak (mutism). In other cases again there are recurring spasms of the respiratory muscles, leading to the utterance of curious coughs, cries and grunts; while in some there is obvious mimicry, as of the bark of a dog. Anæsthesia is rare, but not so hyperæsthesia. Headache with superficial tenderness of the scalp is not uncommon, and other skin areas may be exquisitely tender. A combination of tenderness of **joints** with paretic contracture may produce a condition resembling arthritis, and occasionally the joints are swollen as well as tender. The hip is the joint most often affected, apparent shortening of the limb being produced by tilting of the pelvis. Disease of the knee joint also may be mimicked, or there may be marked spinal tenderness with curvature. So also peritonitis or pleurisy may be simulated, but in using that term we must assume that the mimicry is beyond the control of the child's will, though there may exist in consciousness a knowledge that by a stronger effort of the will the disturbance of function would pass away. Further, it must be remembered that the diseased condition may really exist to a very slight degree, the painful sensations accompanying it being greatly exaggerated, and combined with distinct hysterical manifestations.

In some cases the appetite for food is extremely capricious, less

and less is taken, until finally, under injudicious management, all is refused. Of such are the "*fasting girls*," in whom catalepsy is readily produced. Even when food is taken it is often rejected by vomiting, and the patient may be reduced to the most extreme degree of emaciation. It is obvious that in such cases the mental disturbance is so great that the patients must be considered, at the least, on the border land of insanity.

The **diagnosis** of hysteria should only be made after organic disease has been excluded, and it must be remembered that in a case presenting hysterical symptoms there may be slight organic disease, the subjective symptoms of which are greatly exaggerated. In many cases the inconstancy of the symptoms, in distribution and in intensity, will assist diagnosis. In all a full consideration must be given to the surroundings of the patient, and particularly to the psychical characteristics of the mother. If joint disease or abdominal tumour be simulated it may be necessary to anæsthetize the patient, though here also the possibility of the existence of organic disease in a minor degree must not be lost sight of.

The **treatment** should be guided by the view that in hysteria we have to combat an imperfect or perverted nutrition of nerve cells predisposed by heredity to irregular activity. Means should be taken to improve the general nutrition by a sufficient diet contained in meals taken at regular times, by residence in the country, outdoor exercise, and so on, while the child should be removed as far as possible from the influence of fussy and emotional relatives and placed under the control of instructors who will know how to guard against over-pressure and over-excitement. In the poorer classes it is well to enlist the father to exercise the needed firmness and kindness, for the mother is apt to alternate rather than to combine these two essentials. In severe cases hospital treatment often works wonders, for nowhere can that course of treatment which Donkin has happily described as "observant neglect" be better applied. In the richer classes a modified Weir-Mitchell treatment in a suitable home should be recommended. In minor manifestations, such as hysterical cough, a good teacher of elocution who makes the patient breathe and speak on a system will often effect a rapid cure. The routine use of sedatives, such as the bromides, is strongly to be condemned. They should be prescribed only in emergencies, or when there is ground to suspect that fits are sometimes, or in part, epileptic. Tonics, iron, and cod-liver oil are useful adjuncts to treatment by diet and moral suasion.

Pica: dirt eating, earth eating.—The habit of eating earth, plaster, and other indigestible and sometimes disgusting substancest common among the insane and idiots, and with which pregnan, women and hysterical or chlorotic girls are sometimes afflicted is,

occasionally met with in children who are not obviously deficient in intellect.

In some instances the habit begins in early infancy, there is no impairment of general health, and the practice is abandoned at about three years of age. In other cases the practice begins during ill-health at a later age, and disappears when the health improves. It may thus be met with in children suffering from rickets or tuberculosis, or from intestinal disorders, among which must be included the presence of round worms. In such cases the craving for the indigestible articles may recur more than once at varying intervals.

The condition must be regarded as a psychosis. When it commences in infancy, it is as an exaggeration and perpetuation of the natural tendency of the infant to carry every object to its mouth. When the stuff eaten by preference is wall-plaster or chalk it has been supposed that the habit was an indication that the system was in want of lime salt, but no similar explanation can be advanced when the material eaten is coal, mud, or sand.

Infants who indulge in the habit sometimes show great impartiality in the objects eaten—plaster, coal, clay pipes, mud, blacking, sand, cinders, ashes being taken as opportunity occurs. Cachectic children more often confine themselves to a single substance.[1]

The children have a dull, heavy look, an earthy complexion, hollow eyes, and an unhappy expression. The only special symptoms produced are diarrhœa, which is very usually present, and obstruction from impaction of hard masses in the rectum. Dyspepsia is often present, and, if not the cause of the habit, tends certainly to keep it up owing to the gastric uneasiness which it produces.

The **prognosis** is good, as has been inferred above, but in a few cases a fatal issue has been due indirectly to the malnutrition attending the habit of dirt eating.

The **treatment** of the affection must be mainly prophylactic. The child should be kept out of the way of the indigestible objects which it desires to eat, and its mind diverted by suitable amusements. Punishment, as in most other morbid habits, is of little avail, but judicious moral suasion should be resorted to. Efforts should be made to improve the condition of health in general, and of digestion in particular.

[1] Dr. John Thomson, *Edin. Hosp. Repts.*, vol. iii., p. 81.

CHAPTER XXXIX.

TETANY AND LOCAL SPASMS.

Tetany : Etiology ; Symptoms ; Prognosis ; Diagnosis ; Treatment—Local Spasms ; Eyes ; Head and Trunk ; Treatment—Habit Spasms.

Tetany.

Tetany is a nervous disorder characterized by tonic spasms, affecting especially the hands and feet (" carpopedal contractions ") and due, probably, to chronic intestinal toxæmia.

The *age* at which tetany is most frequent is from six months to two years ; it is very rare after five years, though it is said to become rather more frequent about puberty, especially in girls. The *etiology* has been much disputed. The patient belongs, usually, to a neurotic family, and has inherited an unstable nervous system, but, as a rule, the immediate antecedent of tetany is disorder of the digestive functions, though it is observed sometimes as a sequel of an acute infectious disease. Occasionally the presence of *ascaris lumbricoides* appears to be the determining cause. It is more common in winter and spring than in the warmer season, and an attack may be determined by exposure to cold. It has been asserted that tetany is merely a symptom of *rickets*, and Kassowitz has attributed the contractures to irritation of the cortical centres produced by the hyperæmia of bones and meninges associated with cranio-tabes. The connection between tetany and rickets, however, is indirect, and is to be found in the gastro-intestinal disorders so common in rickety children. The most acceptable theory of the pathology of tetany is that under certain conditions of gastro-intestinal derangement, among which gastric dilatation is probably the most important, toxic substances are produced, which, when absorbed, affect the central nervous system. Degenerative changes, probably of inflammatory origin, have been found in some cases in the cells of the anterior horns of the grey matter of the spinal cord (internal part).

The *onset* is preceded by an acute gastro-intestinal attack, or by the aggravation of a chronic disturbance already present, and there is often some pyrexia. Puffy swelling of the backs of the hands is an early symptom. The child cries when moved, the limbs in many cases are kept constantly in one attitude, and passive movements

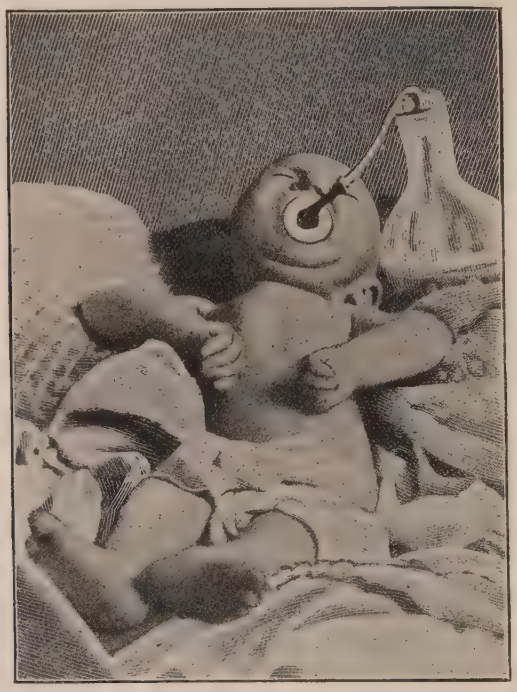
cause pain. The hands are often clenched. In rare cases the first symptom is an attack of general convulsions, or of laryngeal spasm.

The characteristic symptom is *muscular rigidity*, seen first, usually in the hands. Both the flexors and extensors are affected, but the contraction of the former predominates. The hand may assume various attitudes. That most often seen recalls the position of a hand holding a pen. The fingers are flexed at the metacarpo-phalangeal joints, while the phalangeal joints are extended. The thumb is extended and adducted, and the hollow of the palm is deep. In other cases the metacarpo-phalangeal and first phalangeal joints are semi-flexed, while the last two phalangeal joints and the thumb are extended, an attitude which has been compared to that of the hand when drying a sheet of paper on a blotting-pad. In other cases again flexion is complete, the fingers being clasped over the thumb, which is adducted and flexed into the palm. Usually the wrists are rigid and flexed, while the elbows are free, but in some cases the whole limb is rigid in an attitude midway between pronation and supination, with the elbow semi-flexed. The attitude is shown in the drawing (Fig. 64) from a photograph, and has been compared to that of a rider reining-in his horse. The trunk muscles most often affected are the pectoralis major and the trapezius, causing the shoulders to be rigid, or if the trapezius predominate, retracted. The muscles of the neck may also be affected, the head being retracted rigidly. The masseters are contracted in severe cases, the jaws being rigidly closed. Next to the hands the feet are the parts most often involved. Usually there is extreme extension of the ankle with flexion of the great toe. The muscles of the leg are often rigid, but those of the thigh usually escape; sometimes the whole limb is rigid, and rotated outwards (Fig. 64) with the feet in the position of valgus. The contractions are nearly always symmetrical, though they are not always equally intense on the two sides. The affected muscles are firm to the touch, and both antagonizing groups are always contracted simultaneously.

The excitability of the nerves to the galvanic current and to mechanical stimuli is increased. The latter characteristic condition is best observed in the facial. If the finger or the point of a pencil be drawn along the skin from the temple towards the chin, there ensues, if this *facial irritability* be present, a series of contractions in the facial muscles on the same side, best seen in the orbicularis palpebrarum. This symptom is generally present, and may be the only clear evidence to be elicited. A symptom of the same order is *Trousseau's sign*. If the arm be compressed by an elastic band the muscles of the fingers, and sometimes of the fore-arm, pass into the tetanic condition; this is attributed to the mechanical irritation of the nerve trunks by the ligature, but it should be added that the

homologous muscles on the opposite side may also become contracted. Kassowitz has asserted that *laryngeal spasm* is a symptom of tetany, and that its occurrence is pathognomonic. This opinion is too absolute, but it is true that laryngeal spasm occurs with great frequency in tetany, of which, indeed, it may be the earliest symptom. More often the first attack occurs after other symptoms have existed for two or three days. The attacks occur at any time of the day or

FIG. 64.



Attitude, sometimes compared to that of a rider reining-in his horse, which may be assumed in a well-marked case of tetany.

night, may be very severe, in some cases become progressively more frequent and more severe, and have been known to cause sudden death. Arrest of respiration due to spasm of the diaphragm, and of other muscles of respiration may also cause death, which may occur, too, during an attack of general convulsions.

Not only does the number of the groups of muscles affected vary in different cases, but in the same case at different times. There are periods of more or less complete relaxation. When present the tonic

spasms vary in intensity. When at their height, the temperature may be raised 1° or 2° F. The attacks are attended by pain which is aggravated by passive movement, or by pressure. Œdema has been mentioned as one of the prodromal symptoms; it is one of the most constant throughout the whole course of the disease. It is firm and elastic, limited usually to the parts mentioned, but occasionally widespread; its surface is usually pale, occasionally flushed and red. Sometimes irregular areas of erythema are seen on various parts of the limbs and trunk. In rare cases, the sheaths of the tendons on the dorsum of the hand, and possibly also the metacarpophalangeal joints, may be found distended.

The **prognosis** in children is on the whole good. It depends, in large measure, on the character of the gastro-intestinal disorder and the general nutrition of the patient. The more widespread and severe the contractions, the worse the prognosis. In a feeble infant, severe and extensive tetany of the upper and lower extremities, especially if accompanied by retraction of the head, is of bad omen. Death may be due to exhaustion, or, as already stated, to respiratory spasm (laryngeal or diaphragmatic), or to general convulsions.

The **diagnosis** is usually not difficult, though the symptoms of meningitis may for a time resemble those of tetany. In tetany there is no headache, and even if retraction of the head be present, pain is elicited only by movement. The tetanic contractions are symmetrical, the pupils equal, the pulse rapid and regular; vomiting, if present, is not of the cerebral type; the abdomen is retracted, and diarrhœa, instead of constipation, is the rule.

Treatment, if the theory that tetany is due to intestinal toxæmia be accepted, will be directed to the cure of the gastro-intestinal disorder. At first, vomiting should rather be encouraged by giving hot water; but washing out the stomach which has been recommended, is not free from the risk of causing laryngeal spasm. A laxative dose of castor oil or, preferably, calomel should be given and repeated every two or three days. A tetany is sometimes due to *ascaris lumbricoides*, santonin should, in children of over two years of age, be given with the calomel. The diet should be regulated, and pepsin or papaine should be given. At the same time, intestinal antiseptics, such as calomel in small doses (gr. $\frac{1}{10}$), salol, benzonaphthol, naphthalin, or bismuth carbonate or subnitrate should be given in moderate doses frequently repeated. The patient should be protected from cold or excitement, which both tend to produce attacks. A general warm bath is the best treatment for the relief of the painful spasms. Cold compresses applied to the hands and feet will often relieve the rigidity. A weak galvanic current is also to be recommended. The cathode should be placed on the back, and the anode moved slowly over the affected muscles. During severe

attacks, threatening life, chloroform must be given by inhalation. When the symptoms are severe, but less urgent, chloral should be given by enema (gr. iv four times in twenty-four hours for an infant; the dose may be doubled if the desired effect be not produced). Bromide of potassium, of sodium, and of strontium have also been recommended, but do not, as a rule, have much effect. Belladonna is more useful, but must be given in full doses. If the child be the subject of active rickets, advantage will often be obtained by the prescription of phosphorus. The fact that a tetanoid condition follows extirpation of the thyroid gland has led to its administration in tetany. I have not seen very distinct results from this treatment, but Maestro has reported three cases in which recovery took place rapidly; the dose was gradually increased until it reached the large quantity of 30 grains of the fresh thyroid daily.

Local Spasms.

Eyes.—Nystagmus, which is usually lateral, may be a symptom of congenital cataract, or other conditions causing loss or great diminution of vision; of tumors of the cerebellum or pons; of Friedreich's disease, and disseminated sclerosis; of tetany; it occurs sometimes in association with convulsions of an epileptiform nature, as shown by the subsequent development of idiopathic epilepsy; finally, slight inconstant nystagmus may be observed in infants associated in some, but not in all, with error of refraction. The diagnosis of the cause of nystagmus in any case must be made from a full consideration of all the attendant symptoms. Constant nystagmus with wide excursion should be suspected to be due to organic disease.

Head and Trunk.—Clonic spasm of the muscles of the neck causes the head to be rotated, or bent forward, or from side to side. Such movements, which are not uncommon in infancy, begin usually between the ages of six and twelve months. They are rarely seen after three or four years. In *rotary spasm*, the commonest form, the head may be in constant movement, except during sleep, or the movement occurs at irregular intervals, and resembles exactly the gesture of shaking the head in negation. In many cases there is rapid nystagmus, which is increased when the head is held, or may then only be perceptible. *Lateral* spasmodic movements of the head, compounded of slight rotation and flexion on one shoulder, are not very common, and are usually associated with nystagmus. A very similar, but coarser movement may often be noted in children suffering from ear disease. In *nodding spasm*, which is far less common, the head is suddenly flexed forward at intervals, as in the gesture of affirmation, nystagmus is less common, and, when present, has a small

excursion. The prognosis is not good. A large portion of the patients do not survive early childhood, and of those who do many are idiots or feeble minded. *Spasmus nutans* (sometimes called *Eclampsia nutans*), in which at frequent intervals the head and trunk are bowed forward, while at the same time the thighs are slightly flexed, happily called "salaam spasm" by West, is a serious affection. In some cases there is a momentary loss of consciousness, and in others, which have been followed for a sufficient time, it has been proved to be associated with chronic meningitis, or other organic intracranial disease. As is the case with all these spasmodic affections, the movement is sudden and jerky, and must be distinguished from the regular slow rocking to-and-fro of the body on the hips when sitting, which is so common a habit in children with marked rickety deformities, and also from the rapid, sideway, jerking movements of the hips associated with irritation of the anus or genitalia. *Head banging* may be dealt with in this connection. It is not uncommon in children between the ages of six months and three years. It occurs at any time of the day, but most often at night; the child kneels or lies face downwards and bangs its head into the pillow, or the back of the chair. It may go on doing this for hours, unless it hurts its head by hitting it against a hard object. If taken into the arms, it will often continue to hit its head against the nurse's shoulder. It appears to be due in most cases to irritation of the naso-pharynx, ear, or teeth. In acute otitis, the child bores its head into the pillow, but does not bang it. Other children as they lie on the back constantly rub the back of the head into the pillow with a gentle rotatory motion, and in time rub the hair over the occiput quite short. In some cases, no source of local irritation can be discovered, but both the children who bang the head and those who rotate and rub it into the pillow are usually rickety, and the movements are possibly due to irritation connected with rickety changes in the cranial bones. Spasm of the respiratory muscles is referred to on pp. 311–313.

In the **treatment** of these conditions search should be made for some source of irritation with a view to its removal or mitigation. Head-banging or pillow-rubbing should suggest the presence of rickets. In nystagmus the condition of the ocular media should be ascertained. In head-spasms, associated with lapses of consciousness, the condition should be regarded as epileptic and should be treated accordingly. In head-nodding, or rotation without lapses of consciousness, it appears to be justifiable to treat the case as one of chronic meningitis, although it must be confessed that marked results are hardly to be expected, although in some cases the movements cease eventually.

Habit spasms, or *convulsive ties*, of various kinds are common in children, and may persist after childhood. In some cases the manifestations are hysterical, or there is at least an hysterical element.

Others are habits, such as sniffing or clearing the throat, persisting after the cause has ceased. Some of the severer forms are associated with mental defect, which may amount to idiocy.

Spasm is most common in the facial area. Sudden contraction of the orbiculares palpebrarum is often observed. Occasionally this spasm is confined to one side, but is then usually associated with spasm of other muscles of the face on the same side, and occasionally with spasm of the sterno-mastoid. Or the muscular spasm which causes flexure of the head with some rotation is associated with spasm of the lower face muscles only, so that the patient looks as if he were perpetually settling his collar, and in some cases the trick does arise in connection with the wearing of tight bands round the neck. In other cases again the facial spasm is associated with contraction of the scalp muscles, causing the hairy scalp to be shifted backwards and forwards, or the platysma on one or both sides is affected either alone or in association with the facial muscles. The movements in habit spasm of all kinds are short and sudden, and are usually repeated several times in series at irregular intervals. Groups of muscles of the trunk are occasionally affected, causing sudden movements ("electric chorea"), or the distribution may be such that the movement is apparently purposive (*e. g.*, "saltatory spasm"). In such cases the deep reflexes are usually exaggerated.

Associated with the muscular spasms, or, in some cases, without any very obvious habit spasm, curious mental disturbances may occasionally be met with. Thus the child may take to uttering suddenly, and without rhyme or reason, interjections such as "Ah!" or cries of alarm—"Fire!" or "Murder!"—or oaths and foul expressions (so-called *coprolalia*); or may repeat the same word over and over again (*echolalia*); or ask the same question with maddening iteration (*manie de pourquoi*); or, before performing ordinary actions of life, such as putting on its boots, must go through some curious antic, or repeat some sentence as though it were an incantation: or it must count up to a certain number (*arithmomania*); or, again, it has an imperative desire to touch certain objects, so that it cannot leave a room without placing a finger on certain pieces of furniture (*manie de toucher*). As to the treatment of these odd tricks, it is not easy to lay down any general rules. It is necessary to attempt to stimulate the will of the patient, and to induce him to exercise voluntary control over the movements. The condition is often related to hysteria, and educational and other treatment of the kind indicated for that condition is of use also for the correction of habit spasms.

CHAPTER XL.

ECLAMPSIA AND EPILEPSY.

Infantile Convulsions: Causes; Symptoms; Treatment—Epilepsy: Etiology; Symptoms; Jacksonian Epilepsy—Diagnosis of Epilepsy and Eclampsia—Prognosis and Treatment of Epilepsy.

Infantile convulsions (*eclampsia*).—Convulsions, varying as to extent and the parts affected, are very common in infancy and early childhood.

Convulsions occurring within a few days of birth are usually due to injury of the brain at birth. They may be one-sided or general, and cease usually after the first fortnight. When observed later they will usually be found to have commenced after the age of six months. The onset of structural disease of the brain or its membranes may also be attended by general convulsions; but, with these exceptions, the **etiology** of infantile convulsions is obscure. The development of the nervous system is not complete at birth, and its functions are not organized fully until a much later date. The lower centres are organized earlier than the higher, and we may suppose that, wanting effectual inhibitory control from above, they are more prone to excessive action in response to peripheral stimuli.

Rickety children are more liable than others to suffer from convulsions. The nervous centres share in the defective and perverted nutrition, which is the underlying cause of all rickety phenomena, while gastro-intestinal disorders, themselves capable of determining convulsions, are very common in rickets. In other cases the unstable condition of the nervous system is associated with a neurotic family history.

It may be impossible to discover any cause, but in a large proportion of cases some recognized source of toxæmia is present, and when it ceases the fits disappear. To this category belong the convulsions which occur at the onset of febrile diseases, or during the course of acute or chronic gastro-intestinal affections. In other cases, some source of peripheral irritation appears to be the determining cause, since the fits cease when it is removed. Among such sources of irritation must be counted indigestible food, or large masses of undigested food in the stomach, worms or fæcal accumulations in the intestines, disturbed dentition, otitis, and phimosis.

Falls and blows on the head also seem to determine general convulsions in some cases. The convulsions which occur as a complication of bronchitis, and in rare cases attend the paroxysms of whooping-cough, are attributed to cerebral congestion.

The *fits* vary greatly in extent, severity, and duration. If attacks of respiratory spasm (see p. 311) be excluded, it may be asserted that infantile convulsions are attended by loss of consciousness in nearly all cases. Often no premonitory symptoms are noticed, but in other cases the child has been restless with twitching of the arms and grinding of the jaw. Suddenly the arms and legs become stiff, the eyes fixed and staring, or rolled up under the upper lids, respiration is arrested, the head is retracted, and finally the whole body becomes stiff. This stage of tonic spasm is usually followed by clonic convulsions, more or less severe and prolonged, affecting the upper and lower extremities, face and eyes. If the tonic stage is brief, the clonic convulsions slight and few, the whole fit may last less than a minute, and after lying in a drowsy state for a few minutes more the child sits up and appears little the worse. In severe cases the clonic stage is succeeded by a period of drowsiness or stupor, the length of which is in proportion to the severity and duration of the seizure. Fits may succeed each other at short intervals, the patient may then become comatose, and die in the course of a few hours.

The **prognosis** depends partly on the severity of the seizures, but mainly on the frequency with which they recur. A large proportion of the deaths certified as due to convulsions ought more correctly to be assigned to the gastro-intestinal, or other diseases of which they are complications. On the question of recurrence it is in my opinion quite impossible to speak with any confidence, unless the convulsions have occurred at the onset of an acute febrile disease, in which case a hopeful prognosis is justified. If, after the removal of some source of irritation, the fits do not soon recur, the prognosis is good. If the fits have already recurred several times, the chance of repetition is great, since a kind of habit becomes established. Finally, the possibility that the infantile convulsions are the beginning of epilepsy must not be ignored.

The **diagnosis** will be considered later, under Epilepsy. One-sided fits, or those followed by paralysis, are most probably due to organic brain disease.

In commencing **treatment** attention should be directed to the discovery of a source of toxæmia or peripheral irritation. If the temperature be high the probability that the convulsions mark the outset of an acute febrile disease should be considered. If indigestible food have been swallowed recently, or if the fit have been preceded by nausea, the stomach should be emptied by an emetic, or by washing

out. If there be a history of colic, or recent acute diarrhœa, calomel should be given by the mouth, and a copious injection by the rectum. If the temperature be high the injection should be about 85° F.; if there be little or no pyrexia, and in any case if the infant be feeble, at 97° or 98° F. Subsequent injections may be medicated by the addition of antipyrin (gr. iij–iv at one year), which is a useful sedative as well as an antipyretic. Chloral is a remedy which should be reserved for severe cases. If, however, the convulsions be long-continued and oft-repeated a little chloroform should be given by inhalation, so that time may be gained for other measures. A simple warm bath at about 92°–94° F. has a valuable sedative effect; if the body temperature be high, cool affusions to the head during the warm bath may be used; or the temperature of the bath may be made a little lower. If after the convulsions have ceased congestion of the face and conjunctivæ persist, a warm pack to the legs, or to the lower limbs and trunk also, should be given. [A mustard-bath may be given. Mustard-bath—mustard, 1 oz. to each gallon of water (at 98° to 106° F).] If the child is much depressed, brandy or eau de cologne should be added to the water. No advantage can be expected from scarification of the gums as a matter of routine, but if the mucous membrane be stretched by a tooth near the surface, an incision down to the tooth will remove one source of peripheral irritation, which is very real, though its frequency and importance as a cause of eclampsia has been greatly exaggerated. The after-treatment should be systematic. At first bromides should be given, or if there be stomatitis or painful colic, opium in small doses for a short time. The diet should be regulated, imperfect digestion or gastro-enteritis combated, rickets treated, and the child should be placed under the best available hygienic conditions. If convulsions recur, it is advisable, while still giving attention to possible sources of irritation at the periphery, to institute a course of bromides.

Epilepsy.—If epilepsy be defined, with Osler,[1] as "an affection of the nervous system characterized by attacks of unconsciousness with or without convulsions," and it would be difficult to frame another definition less open to objection, then it follows that no hard and fast line can be drawn between infantile eclampsia and epilepsy. Indeed, v. Strümpell[2] goes so far as to write that "the epileptic attacks of infants during the first year are commonly called eclamptic," and admits only the distinctions that in the imperfectly developed brain of the child an epileptic explosion is much more easily induced, and that the prognosis is much better.

[1] Osler, "Prin. and Prac. of Med." Second Edition, 1895, p. 1002.

[2] Penzoldt and Stintzing's "Handbuch d. Spec. Ther.," Bd. v., Abt. viii., p. 454.

Gowers[1] found that the ages at which the largest number of cases of epilepsy commenced were fourteen, fifteen, and sixteen years, that one-fourth of the cases commenced before ten, and one-eighth before three. Hasse[2] states that in 9 per cent. the fits began so soon after birth that the epilepsy might be called congenital. The influence of heredity is shown by the fact that Gowers[1] found that in one-third of his cases of all ages there was a history, in ancestors or collaterals, of epilepsy or insanity. It is brought out well also by Echeverria's[2] statistics of 135 families in which either the father or mother was epileptic; there were 554 descendants, of whom 246 died early, 203 suffered from epilepsy or other nervous disorder, and only 105 were healthy.

The epileptic fit is due to an abnormal discharge in the higher (probably cortical) cerebral centres, which affects a larger or smaller number. Such discharges may be provoked by organic disease (tumour, abscess, etc.) or by trauma. In such cases, which are discussed elsewhere, the convulsion may be limited to the corresponding side of the body. In cases not due to gross organic disease of the brain, the determining cause of the fits is not as a rule discoverable, and to them, therefore, the term idiopathic epilepsy is commonly applied.

In cases which begin in infancy the conditions already mentioned as *causes* of infantile eclampsia must again be invoked. Peripheral irritation seems to be in some cases the exciting causes of epilepsy,[3] while in a larger number it renders the fits more severe and more frequent. Blows and falls on the head which leave no definite evidence of organic disease may yet be followed by epilepsy, which is also an occasional sequel of exposure to the sun. Toxæmia, from one source or another, must be looked upon as the determining cause of the initial fits in many cases. It will account for those, not due to gross lesion, which arise in the course of acute infectious diseases; scarlet fever, measles, and typhoid fever, especially the first named. Again, various conditions of the digestive organs appear to be the determining cause in some cases; thus the fits may date from an attack of gastro-enteritis. Again, in many epileptic children the bowels are constipated, owing apparently to "torpidity" of the liver, which is enlarged; the face has a heavy expression, the lips and tongue are congested and there is tenderness, sometimes pain, in the

[1] Gowers, "Dis. of Nerv. Syst.," 1888, vol. ii., pp. 676 *et seq.*

[2] Baginsky, *Lehrb. d. Kindrhlde.*, Vfte. Auf. l, 1896, S. 539 *et seq.*

[3] In Burchard's case, for instance (*Arch. of Ped.*, 1895, p. 35), a boy began to have epileptic convulsions, so diagnosed by, among others, Charcot, at the age of ten. When he had reached the age of twelve it was noticed that he had a long adherent prepuce and discharge from the urethra. Circumcision was performed and much retained smegma removed. He had a fit on the first and second days after the operation, but not another down to the date, sixteen years after the operation, when the report was made.

hepatic region. Idiopathic epilepsy occurs occasionally as a complication of inherited syphilis without other discoverable cause. The fact that a large number of cases begin at or about the age of puberty suggests that the changes in the nervous system which attend the development of the sexual organs have some influence, and it should be noted that during the first three decades of life there is a preponderance of female cases, especially marked in the second decade, but still noticeable in the third. Masturbation, especially in boys, is a frequent concomitant of epilepsy, but whether it should be regarded as a cause or as a common consequence of a remoter cause is open to doubt. Sudden emotions, especially fright, are held to be capable of producing epilepsy. A fit occurring immediately after the emotion is probably hysteroid, but true epileptic fits occasionally occur after an interval. Finally, it may be said that there is no cause of an epileptic attack so potent as the condition of nervous system left by a previous attack, so that, once established, the fits may continue long after the exciting cause of the first has been removed.

The **symptoms** of epilepsy in children do not differ from those in adults and need not here be described at length. Children are liable both to the grave and to the minor form (*petit mal*). It is probable that the proportion of cases of *petit mal* and of those in which the fits occur in groups is greater in children than in adults. As Henoch has pointed out, it is not uncommon to meet with cases in which a child has for years experienced "peculiar sensations" at irregular intervals. Finally, the occurrence of an epileptic fit after one of these "sensations" shows that they have been auræ. In some, at least, of these cases the "peculiar sensation" is followed by a loss of consciousness, so short that it will not be noticed unless looked for. The seizures of minor epilepsy are often described as "faints." The child sinks back in its chair or falls to the ground, the face becomes pale, and consciousness is lost. In a moment or two it recovers consciousness, but is drowsy. Urine may be passed during the fit, and if minor attacks occur at night, occasional nocturnal enuresis may be the first symptom to arouse suspicion, which will be confirmed if there be now and then unusual drowsiness in the morning and purpuric spots about the neck, or if the tongue be found bitten. The recognition of the real nature of such cases is important, not only as regards treatment, but also because some patients who suffer from nocturnal fits have a tendency to turn over on the face and so run the risk of suffocation. Some attacks of minor epilepsy, especially in girls, are followed by hysteroid convulsions. This statement would not be accepted by Charcot and his followers, who hold that such fits are from the first hysterical, though they admit that the hysterical fits may alternate with the epileptic fits and

thus lead to a mistaken diagnosis. In some patients vomiting follows the fits, and is a source of danger, as food may be drawn into the larynx. Automatic actions immediately after the fits, such as undressing, climbing upon furniture, or sudden motiveless assaults upon others, are observed in some cases in children as in adults. After a severe fit there may be temporary muscular weakness amounting to actual paresis, hemiplegic or paraplegic in distribution, corresponding to the parts most affected during the fit. The mental condition of epileptic children varies very greatly. As a rule they are backward, but if the fits be neither frequent nor severe there may be little or no obvious deterioration, at any rate for some years. If the fits be frequent or very severe the child is dull and depressed, and it is difficult to teach because the memory is, as a rule, defective. In other cases the child, during the interparoxysmal periods, is noisy, irritable, and mischievous, subject to outbursts of temper, and shows great lack of self-control, and in some cases definite moral perversions. In the most severe and progressive cases of epilepsy dementia ensues sooner or later.

Jacksonian, or focal epilepsy, in which the spasms commence in a small area, usually one of the limbs, is symptomatic of organic irritative disease of the cerebral cortex (tumours, abscess, etc., *q. v.*). The convulsive movements beginning in one limb may be limited to it, may extend to the other limb of the same side, or may become general. Except in the alternative last named the fits are often, perhaps usually, unattended by loss of consciousness. Infantile hemiplegia, the onset of which is usually attended by convulsions, may be complicated at a later date by epilepsy. If the paralysis have almost cleared up it may escape discovery, especially if the fits, as happens not infrequently, only recur with severity about the age of puberty.

For the **diagnosis** of idiopathic epilepsy the point of primary importance to be recognized is that the fits are recurrent. As has already been observed, no hard and fast line can be drawn between infantile convulsions and epilepsy, for, as Gowers has said, "whenever attacks continue after their cause has ceased, the condition is inseparable from epilepsy." It must be remembered that epileptiform convulsions even in very young children may be uræmic, and that convulsions from this cause may be one-sided. The epilepsy due to gross lesions will be distinguished by the history, the mode of onset of the attacks, their limitation in area, and by the recognition of other symptoms of organic disease—headache, vomiting independently of the fits, optic neuritis, paralysis. In epilepsy having its origin in infantile hemiplegia careful examination will reveal the presence of paresis, rigidity, and exaggerated reflexes on one side. Definite hysterical convulsions do not occur in infancy, are very rare

in childhood, but become more frequent at puberty. They may be distinguished by noting that they are generally induced by emotion; that the movements, which are struggling or purposive, irregular in distribution, and often continued with partial intermissions for as long as an hour or longer, are generally accompanied by crying and whining; that the face is flushed; that the pupils react normally; and that if there be loss of consciousness it does not continue throughout the whole fit. Fits which begin quite suddenly, with pallor of the face, fixed pupils, and loss of consciousness, even if these symptoms be followed by hysteroid symptoms, should be regarded as epileptic.

The **prognosis** of epilepsy commencing in childhood is worse than in adults. Fits which cannot be distinguished from those of epilepsy may, when they occur under two years of age, disappear; but if definite recurrent attacks are observed after five or six years of age, the prospect of cure is small, though improvement may take place under treatment. Immediate danger to life is less than in infantile eclampsia, though sudden death is caused occasionally by suffocation in bed, or by the impaction in the air passages of food rejected from the stomach immediately after the fit.

In the **treatment** of epilepsy, it is especially important in young children to search for one of the peripheral sources of irritation or of toxæmia mentioned above. Neuralgia, dyspepsia, intestinal worms, phimosis, rickets, and anæmia should receive appropriate treatment. When constipation is present, saline aperients are indicated, and their use may suffice materially to reduce the number and severity of the attacks. If the liver is enlarged, ammonium chloride should be given systematically in full doses. In every case, in fact, it is important to endeavor to improve the state of the general health, in whatever respects it may be found defective, and not to trust alone to the routine administration of bromides. Potassium bromide is the most efficient of these, and should be given at once in full doses. If the fits cease, the drug should not be stopped, though the dose may be reduced. Premature arrest of a course of bromide is followed, almost invariably, by recurrence, and as a rule it is not prudent to withdraw the drug altogether until the patient has been free from fits and "sensation" for several years. The object is to give at first as large a dose a possible, short of producing the symptoms of bromide poisoning, which are lethargy, physical depression, muscular weakness, cold extremities, and feeble pulse. Impetigo occurring as a consequence of bromide ingestion should be treated by attention to cleanliness, by antiseptic ointments, and by the prescription of small doses of arsenic combined with the bromide. True bromide rash, which is, however, rare, may render it necessary to suspend the treatment. The drug may be given in three doses after

meals, or, if the fits are usually nocturnal, a double dose, or half the daily quantity (at first gr. xxx, increasing to gr. lx for a child of eight), should be taken in the evening. Gowers recommends large doses, taken in half a pint of water, at intervals, at first, of two days, gradually increased to four days, the doses being correspondingly increased; both are then decreased, so that the whole course lasts six weeks. Subsequently, small daily doses should be given. When the drug is given in the ordinary way, the dose may be reduced cautiously when the severity and frequency of the fits have distinctly declined. Sodium and ammonium bromides are preferred by some, under the impression that they are less depressing than the potassium salt. In some cases in which potassium bromide fails to produce much effect, the addition of potassium iodide, as recommended by Brown-Séquard, is followed by rapid improvement. Digitalis may be combined with potassium bromide with advantage if there be cardiac dilatation and feeble circulation, and Gowers states that it is of use also in nocturnal epilepsy. Belladonna, which is well borne by children, is useful sometimes, but opium is not to be recommended. Borax, which has been strongly recommended as an alternative for the bromides when these are not well taken, has never in my hands had a favorable effect in children. In cases with a prolonged aura, the inhalation of amyl nitrite has been known to prevent the threatened attack, and when the aura begins definitely in the hand or foot, a ligature round the limb has had the same happy effect. It would be useless to enumerate all the drugs which have been used with alleged benefit in epilepsy; our main reliance must be upon the bromides coupled with an intelligent treatment of any concomitant symptoms. During the fit, little can be done beyond taking means to prevent the patient from injuring himself, especially from biting the tongue. Nothing should be given by the mouth, and, after the fit, the child should be allowed to sleep.

An epileptic child should lead a quiet, regular life, free from excitement. It should be educated either alone, or in a special class for backward children, or, and this is probably the best course, it should be entered in an epileptic colony.

[The surgical treatment of epilepsy has attracted much attention, but, except in a few limited cases, cannot be said to offer much encouragement. It should, of course, be considered only in those cases of "partial epilepsy" pointing to a definite focus of disease. Sachs advises that in case of brain injury the damaged tissue should be removed. If allowed to remain he considers such tissue a constant menace to the patient.]

CHAPTER XLI.

MENINGITIS.

General Etiology and Symptoms : Intracranial Tubercle ; Tuberculous Meningitis—Anatomy—Etiology—Symptoms—The Diagnosis of Meningitis—Treatment—Posterior Basal Meningitis—Hydrocephalus.

Meningitis.—The commonest *cause* of acute meningitis in childhood is tuberculous infection, but it may occur in the course of other specific infections—small-pox, scarlet fever, measles, enteric fever, pneumonia. The symptoms formerly attributed to rheumatic meningitis are more probably due to hyperpyrexia. Next to tubercle, injury to the bones is the most common cause of acute meningitis in childhood. Otitis may lead to meningitis, but more often determines inflammation of the substance of the brain or sinus-thrombosis. Meningitis may be an incident of general septicæmia, or pyæmia, and in some cases the peritoneum, pleuræ, and meninges are affected simultaneously with purulent inflammation, the primary seat of which cannot be ascertained.

Acute meningitis may be limited to the base, as is usual in tuberculous meningitis, or to the convexity and superior longitudinal sulcus, as is sometimes the case in specific diseases, but when purulent, it almost invariably quickly becomes general. Cases in which the inflammation is limited mainly to the posterior fossa will be considered separately.

Certain **symptoms** are common to all forms of meningitis. The order in which they appear varies, but, as a rule, the earliest is the so-called cerebral vomiting, in which the contents of the stomach are ejected suddenly, without evidence of nausea, and either without relation to meals or without connection with either undue quantity or improper quality of the food. Headache, persistent, but subject to exacerbations, is often an early symptom and persists after delirium has come on. The delirium, which is attended by drowsiness, is, at first, a mere wandering at night, with some eager garrulity by day. It is generally quiet, though the speech is often hasty and the tone anxious. Later it becomes muttering and almost continuous, until it is replaced by coma. General convulsions are frequent in infants and young children. Paralysis, or more usually paresis of cranial nerves, may develop slowly or rapidly, and may disappear and re-

appear several times. The limbs may be weak, paralyzed, or rigid. The convulsions sometimes, and the palsy and rigidity, perhaps, as a rule, are hemiplegic in distribution, though complete hemiplegia is not common.

In purulent meningitis the temperature rises quickly, with or without rigors, to 103°–105° F. In other forms it may not exceed 100° F., or may even be subnormal until just before death, when hyperpyrexia may occur. The pulse may be frequent throughout, or after being frequent it may become slow and irregular for a time, and then again shortly before death, very rapid. Respiration is not, as a rule, quickened in proportion to the pulse. In the last stage it may become irregular, with long pauses, or be distinctly of the Cheyne-Stokes type.

Intracranial tuberculosis.—Tuberculous lesions within the cranium occur under two forms : tuberculous meningitis, which, in its several symptoms, resembles meningitis from other causes ; and solitary tubercle, which produces symptoms of the same nature as those caused by cerebral tumors generally.

Tuberculous meningitis is the commonest form of fatal cerebral disease in children. The extent of the tuberculous lesions found after death varies greatly, and frequently the point of entry of the infection cannot be discovered. In some, perhaps the majority of cases, the infection of the meninges is a part of general tuberculosis, the spleen, the lungs, and the serous membranes generally being infected ; this is especially the case in the youngest children. In others, the meningitis is clearly secondary to some local tuberculous infection of the bones, or joints, or of the bronchial, cervical, or mediastinal glands. In others, again, the meningitis appears to be the primary lesion, tubercle being plentiful in the cerebral and spinal meninges, but in other organs absent or scanty.

Etiology.—F. Brandenburg[1] estimated from a study of the cases at the Children's Hospital in Basle that tuberculous meningitis constituted 8 per cent. of all the cases of tuberculosis occurring in the first year of life, 15 per cent. of those occurring in the second year, 4 per cent. of those in the third, and 37 per cent. of those in the fourth year of life. He came to the conclusion that in 34 per cent. of the cases of tuberculous meningitis the infection may have been derived from some other member of the family. This is about the same proportion as for tuberculosis generally. In two-thirds of the cases the bronchial glands were found to be caseous, indicating, probably, that the infection had found entrance by the air passages. Among the determining causes, blows on the head and injuries of bones already the seat of tuberculous disease must be reckoned. Brandenburg found that in 8.3 per cent. of the cases of tuberculous

[1] *Jahrb. f. Kinderhlkd.*, Bd. xxxii., S. 159.

meningitis the patients had been suffering from tuberculous disease of bone which had not been operated on; while in 17 per cent. it followed operations on bone disease.

Accepting the view that the infective principle is derived from the air or food, we may look for predisposing causes to insanitary conditions, especially overcrowding, with its attendant evils, including imperfect ventilation; to that hereditary proneness to tuberculous infection which constitutes the tuberculous diathesis; and possibly to intellectual over-strain and worry, though the importance of causes of this nature has probably been exaggerated.

The disease has occurred at the age of six weeks, but it is not common in infancy. It is more often seen in the second year of life, and is commoner between the ages of two and ten years than at any other age.

Pathological anatomy.—Tuberculous meningitis is essentially a basal meningitis, determined by the formation of tubercles in relation with the blood-vessels. Frequently ventricular effusion is superadded.

The tubercles, which are usually found in greatest number in the fissure of Sylvius, are scattered along the vessels in decreasing number from the base upwards, and may be encountered in the margins of the superior longitudinal fissure. They vary in size with their age, and at the base the attending fibrinous effusion may be so considerable as to mask them. The tuberculous process appears to begin in the perivascular space, and to extend along the arterioles into the brain substance; in any case there is always some cerebritis, whether secondary to the meningitis produced by the meningeal tubercle or set up by the tubercle around the vessels penetrating the brain substance. In the ventricle, tubercle, if present, is to be found in the choroid plexus; it leads to effusion into the cavity of the ventricles with attendant softening of the surrounding cerebral substance. To the frequency with which an excess of fluid is found in the ventricles, and to the not uncommon existence of very copious effusion, the affection owed the term "acute hydrocephalus," formerly often applied to it.

Symptoms.—The nature of the earliest recognizable symptoms varies according as the affection of the meninges is the first, or at least the most rapidly developing tuberculous lesion, or whether it is only a part; perhaps a late part of a general infection.

In primary tuberculous meningitis it is customary to describe prodromata and three stages of the developed disease, though the symptoms of the various stages are far from constant.

In the *prodromal stage* certain slight deviations from health accompany the commencing infection of the meninges. The child loses flesh, the bowels are irregular, the appetite is capricious, and

sleep disturbed. There are momentary attacks of dizziness, or lapses of consciousness; the character changes and the child becomes dull and heavy, or alternates between emotional excitement and drowsiness.

The *stage of invasion* ensues in a few days, or after a week or two, or even longer. The characteristic symptoms of this stage are: (1) headache, referred generally to the front or top of the head, and persistent, but liable to exacerbations; (2) vomiting without relation to meals; (3) obstinate constipation; and (4) irregular, sighing respiration, especially if present during sleep. These symptoms in a child who has been for some weeks failing in health and altered in disposition should lead to a careful examination for other symptoms: these are fever (100° to 101° F.) at night, rapid pulse, areas of hyperæsthesia, vaso-motor instability, avoidance of noise and light, tenderness of the eye-balls, and somnolence. Certain other symptoms may exist from an early stage. Of these, two, if present, are extremely characteristic. The one is a striking loss of elasticity of the skin, so that a fold pinched up by the finger and thumb only slowly disappears; the other, a peculiar, soft condition of the abdomen, which gives to the hand a feeling precisely like that of a bag of dough. Another symptom is slight unsteadiness of the trunk in standing (static ataxy). Rigidity of the muscles at the back of the neck and retraction of the head are early symptoms when the posterior fossa is the area affected, or mainly affected.

The *stage of irritation* follows usually in about a week, and lasts three or four days. It may be attended by a fall of temperature, while the pulse becomes slow and intermittent, though easily quickened by exertion. The respiration also is irregular, and may be distinctly of the Cheyne-Stokes type. The headache becomes more severe, and may lead to the utterance of a high-pitched cry at irregular intervals. Grinding of the teeth and chewing motions of the jaws are often present. The child is quietly delirious or somnolent, lying on its side with eyes nearly closed, brows contracted, and the knees flexed on the abdomen; the back is also flexed, and when to this is added retraction of the head, the appearance is very striking. Even in this condition it may be aroused for a moment to answer a question, though it resents such interference. The belly remains soft, but is retracted (the boat-shaped, or canoe-shaped belly). The vaso-motor instability is marked. Stroking the skin with the finger is followed by the gradual development of a bright red streak which lasts for some time (*tâches cerebrales*, "cerebral flush"). During this, but sometimes at an earlier stage, affections of the cranial nerves may be observed—dilated and unequal pupils, strabismus, partial ptosis, imperfect closure of the eyes, slight facial paresis. Optic neuritis may exist, and choroidal tubercles may be discoverable.

The *stage of coma* sets in gradually; the temperature rises and

may attain 104° or 105° F.; the pulse also becomes more rapid, and respiration more irregular. It ceases to be possible to arouse the child; the tongue becomes dry, the lips cracked, the cornea obscured by muco-pus. Convulsions, general or partial, may occur, or opisthotonos, and the limbs on one side (seldom one limb only) may be paralyzed, while the ocular paralysis grows more marked. Death may be determined by exhaustion, hastened by the formation of bedsores, the accumulation of mucus in the chest, or by a convulsion.

Diagnosis of meningitis.—Certain acute specific diseases, especially enteric fever, pneumonia, ear disease, and the general condition produced by some intestinal disorders, are liable to be confounded with meningitis, and in many instances a confident diagnosis cannot be made until after the case has been under observation for some days. As a general rule, it may be said that the diagnosis of meningitis should not be made until all other conditions which may produce similar symptoms have been excluded. This observation applies more especially to tuberculous meningitis. In acute suppurative meningitis, a probable cause is commonly to be discovered, and the symptoms are so acute that there is little room or time for hesitation. The mistake most often made is to attribute symptoms due to intestinal disorder to meningitis, and next to that to diagnose tuberculous meningitis when the disease is really enteric fever. In doubtful cases the serum test will in future be of great assistance. [Lumbar puncture is also of great aid in diagnosis, see p. 126 and below.] The resemblance between the earlier symptoms of tuberculous meningitis and enteric fever is, indeed, often very close, and it is open to question whether the latter does not in some cases determine the onset of tuberculosis. In enteric fever, the headache may be severe at the onset, and there may be constipation; while in tuberculous meningitis there may be diarrhœa from intestinal ulceration, and abdominal tenderness due to mesenteric disease. Sir William Jenner has insisted on the diagnostic value of the observation that the headache of enteric fever subsides with the onset of delirium, while it persists with delirium in meningitis. But this valuable criterion may fail us in infants and young children. In infants, the conditions corresponding to delirium is characterized by restlessness, sudden screams, and an expression of fear. The sudden onset of high temperature and headache followed by delirium, or the condition just described, should suggest in infants pneumonia, and in older children either pneumonia or enteric fever. The pyrexia of enteric fever is more regular than that of meningitis, while the pulse is rapid and does not present the irregularity or slowness so often observed in meningitis. If pneumonia is developing, even though no physical signs can be detected, the pulse-respiration ratio will almost certainly be disturbed, and there will be respiratory movements

of the alæ nasi. The inelastic skin and soft doughy belly of tuberculous meningitis are not observed in any other condition. Ophthalmoscopic examination may reveal choroidal tubercle or optic neuritis. The former renders the diagnosis of tuberculous meningitis certain; the latter makes it probable, since optic neuritis is rare in acute specific diseases, and only occurs in a late stage. The absence of changes in the fundus is of no value either way, since choroidal tubercle is comparatively rarely to be observed during life.

It is said that ear diseases may produce optic neuritis without the intervention of meningitis, and whether this be so or not, it is sometimes difficult or impossible to decide whether the general symptoms which attend acute otitis are due to that condition alone or partly to complicating meningitis. All that can be done under such circumstances is to ascertain by paracentesis of the tympanum if pus is present in the tympanic cavity, to evacuate it if found, and to watch the effect of treatment directed to the relief of the local conditions. If the acute otitis involve both ears simultaneously, the resemblance is the greater, and the difficulty is practically insuperable. A similar difficulty may arise where chronic tuberculous disease of the petrous bone induces general symptoms; unless choroidal tubercles can be seen, or tubercle bacilli discovered in the fluid withdrawn by Quincke's lumbar puncture, a confident diagnosis of tuberculous meningitis may be impossible.

[There can be no doubt of the diagnostic value of lumbar puncture in cases of meningitis. Normal spinal fluid is always absolutely clear; that from a case of meningitis invariably cloudy. The cloudiness may be slight and for detection may require shaking in the test-tube and comparison with another test-tube of clear water. It is due to the presence of cells, the character of which varies with the variety of the meningitis. After withdrawal of the fluid fibrin is formed, and Wentworth considers these cells and fibrin pathognomonic of inflammation in the meninges. Albumen is also present in increased amount, from $\frac{1}{30}$–$\frac{1}{10}$, of one per cent., normal spinal fluid containing from $\frac{1}{60}$–$\frac{1}{50}$ of one per cent.

The following micro-organisms have been demonstrated in spinal fluid withdrawn by lumbar puncture from cases of meningitis, either by microscopic examination of the sediment, by cultures, or by inoculation experiments: the diplococcus lanceolatus, staphylococcus pyogenes aureus, pneumococcus, diplococcus intracellularis meningitidis[1] and bacillus tuberculosis. The last named is often difficult to find in cover-glass preparations of the sediment and inoculation experiments are sometimes necessary to demonstrate its presence.]

Infants and young children exhausted by diarrhœa or other depressing disease may pass into a condition of nervous collapse to

[1] Wentworth, *loc. cit.*, p. 127.

which the terms "spurious hydrocephalus" and "hydrocephaloid" have been applied. The child is somnolent or comatose, the breathing shallow, the abdomen soft, and the limbs relaxed. It will be noticed, however, that the fontanelle is depressed, the abdomen, though lax, is not doughy, there is no elevation of temperature, the pulse is regular and often fast, and there is no strabismus or definite paralysis of the limbs. It must be added, however, that in a few rare cases strabismus and the general symptoms above noted have been known to pass away after the passage of a round worm, and, further, that retraction of the head may exist along with the symptoms of spurious hydrocephalus. The diagnosis, in such cases, must depend upon careful observation of the case, and of the effects of treatment. In older children, lethargy, constipation, and even strabismus, may be due to hysteria (*q. v.*); but there is no pyrexia, the pulse is regular, the strabismus is convergent, and the pupils small. Retention of urine or the passage of large quantities of pale urine at irregular intervals may be taken to confirm the diagnosis of hysteria; but, on the whole, the danger is far greater of ascribing the early symptoms of tuberculous meningitis to hysteria than of falling into the converse error.

The **treatment** of acute meningitis, whether suppurative or tuberculous, offers little hope. The patient should be put to bed in a quiet, shaded room, an ice-bag applied to the head, and warmth to the feet; a brisk purge should be given, and a gentle laxative effect kept up by the exhibition of salines. Nourishment must be administered systematically at short intervals, the stomach-tube being used if the child refuses food. The value of a mercurial course is open to doubt; it is, of course, out of the question in the most acute cases, and not much can be expected from it in tuberculous meningitis; at the same time, if the symptoms be less acute, and especially if they point to posterior basal meningitis, which is sometimes syphilitic, it seems to be justifiable to give mercury either by the mouth or by inunction as rapidly as possible. Even in some tuberculous cases slight temporary amelioration ensues. If the outset has been acute, and if signs of compression are making their appearance, the propriety of draining the excess of fluid from the cranium should be considered before coma has become established. Quincke recommends that puncture should be made in the lumbar region, this part being chosen because the spinal cord ends at the lower border of the first lumbar vertebra. The patient is placed in the sitting posture, and bending forward; the needle of a sterilized syringe is pushed into the canal in an upward direction between the second and third or third and fourth lumbar vertebræ; as much as ʒ iij of fluid may be drawn off slowly. If the case be tuberculous, the tubercle bacillus may be found in this fluid. A large quantity of albumen in the

fluid rather points to the meningitis not being tuberculous. At this stage of the case it will be desirable to consider whether it may not be possible to attempt continuous drainage of the cranium by trephining, and putting a drain, if necessary, even into the lateral ventricle. It seems clear that this treatment has prolonged life even in tuberculous meningitis, and it is possible that recovery may thus be brought about, as in tuberculous peritonitis. ["Freyhan found the tubercle bacilli in the cerebro-spinal fluid of a patient who recovered" —Sachs.]

The **prophylaxis** of tuberculous meningitis deserves special attention. In a child with tuberculous tendency, whether hereditary or acquired, any sign of over-pressure at school should lead to immediate relaxation of studies; the liability to tuberculous meningitis diminishes after puberty, and such children are usually quick and studious, so that they rapidly make up leeway. Attention also should be given to the diet. The appetite is capricious; it will often be found that the amount of food eaten, in particular the quantity of fat is very small. Cod-liver oil is to be recommended, and will sometimes be well borne, even in large doses, when ordinary fatty foods excite only disgust.

Posterior basal meningitis not due to tubercle is met with most often under the age of twelve months, but it is not unknown after that age. It presents a characteristic train of symptoms. Of these, cervical opisthotonos is the most constant, and usually the earliest. The retraction of the head may develop slowly or rapidly, and is accompanied at first by vomiting and irritability, later by stupor. Convulsions may occur at an early stage, but tonic spasm is throughout the dominant symptom. The rigidity may affect not only the muscles of the neck, but also those of the back generally, of the lower limbs, which are rigid in extension, and of the upper, which are rigid in flexion (Figs. 65 and 66). The stupor is associated usually with accumulation of fluid in the ventricles, evidenced by bulging of the anterior fontanelle. Squint is not uncommon, and nystagmus occurs in some cases; but optic neuritis is rare, though some diminution of vision, if not complete loss of sight, appears to be the rule. Slowing of the pulse is much less common than in tuberculous meningitis, but may occur, especially in children over one year of age. The respiratory rhythm is frequently disturbed. Cheyne-Stokes rhythm may occur, but a modification in which the pause is followed by one or two deep inspirations is more usually observed. The abdomen is not retracted, and constipation is not the rule. There may be no fever during the whole time the child is under observation; but if the case is seen from the earliest stage, some elevation of temperature will usually be found to occur then, and in many cases to recur at irregular intervals. Death is some-

times preceded by hyperpyrexia. The course of this form of meningitis is usually long—seldom less than a month, often two or three

FIG. 65.

months. The younger the patient the more acute the course. Some patients recover, though what proportion it is not possible to say; the retraction of the head passes away, and vision and intelligence

FIG. 66.

Posterior basal meningitis, showing the characteristic attitude. From photographs (by Dr. DUDLEY W. COLLINGS) of a child under the care of Dr. Church in St. Bartholomew's Hospital. The patient eventually recovered sufficiently to leave the hospital.

are regained; but some enlargement of the head, doubtless due to hydrocephalus, remains and may be conspicuous for some years.

The **morbid anatomy** of the condition is so far simple that there is in all cases meningitis which begins at the base, and is always most marked in that region. It may remain limited to it, or may extend to the temporo-sphenoidal lobe, or even to the vertex, or downwards into the vertebral canal. It is plastic, and adhesions are very prone to occur both at the base of the brain and in the spinal canal. Hydrocephalus, which is so commonly produced, is due, in most if not in all cases, to adhesions contracted between the cerebellum and medulla, closing the foramen of Magendie or that of Monro, or the aqueduct of Sylvius, obliterating the fourth ventricle, or blocking the posterior arachnoid cistern, or the spinal canal in the cervical region. The fluid by which the ventricles are distended may be turbid and contain fibrinous flakes, but probably only when the inflammatory process is still active. Later it is clear, and consists of pure, or almost pure, cerebro-spinal fluid.

The **etiology** is obscure; traumatism may account for a small proportion of cases; probably, in the majority, the meningitis is secondary to a catarrhal process involving the middle ear. The extension of the infection is no doubt due to micro-organisms. Probably more than one variety is capable of causing limited meningitis in this region, but the varieties have not been identified.

The **prognosis** in any case in which the diagnosis can be made with confidence is bad. Death is the rule, recovery the exception. The more rapid the onset, the greater the probability of a fatal issue at an early date. In such cases the respiration may become suddenly shallow and slow, or irregular. Cyanosis ensues, and the child dies. When recovery does occur it is in many cases complete—the rigidity passes away entirely, vision is regained, and the development of the brain does not seem to be permanently retarded, though the head may remain obviously enlarged, owing probably to persistent ventricular effusion. In other cases chronic progressive hydrocephalus (*q. v.*) ensues.

The **diagnosis** in a well-marked case with constant retraction of the head, irritability passing into stupor and coma, and alteration of the respiratory rhythm is comparatively easy, the only difficulty being to exclude tuberculous meningitis. In the latter, though tonic spasm may occur, clonic spasm, due to involvement of the cerebral cortex, is often a prominent symptom, and there is marked retraction and doughiness of the abdomen. The course is, moreover, generally more acute, and the symptoms more variable. Further, tuberculous meningitis is not very common during the first year of life. On the other hand, there may be considerable difficulty in diagnosis in the early stage. Retraction of the head may be due to tetany, or to

peripheral irritation (see pp. 34, 445). In tetany, however, the tonic spasm is not constant in degree, the hands and feet are usually affected at an early date, and other signs of tetany may be elicited. With regard to middle-ear disease, the question is somewhat different. It is certain that a certain number of cases presenting symptoms suggesting posterior basal meningitis recover after treatment directed to the ear, and if it be accepted that this form of meningitis is usually secondary to otitis media, it may be held that the treatment of the ear disease has cut short a commencing meningitis. If this be admitted, then it would follow that a very considerable proportion of the cases of posterior basal meningitis recover. In all probability, however, otitis media is itself capable of producing more or less marked retraction of the head.

The point is of the less importance to decide because it is desirable to assume that all cases presenting symptoms of ear disease call for immediate **treatment**, whether we hold that the cure of the ear disease has the effect of preventing or of curing the meningitis. Even in the absence of distinct indications of pain in the ear it is, therefore, justifiable to incise the tympanic membrane, since the operation is in itself harmless. If no pus be obtained, and the wound heal rapidly, it will be well to repeat the incision should the symptoms persist. When first seen the patient should be put upon small doses of calomel (gr. $\frac{1}{20}$ to $\frac{1}{10}$ four times a day), which has the double advantage of controlling the diarrhœa so often present, and of producing a mild degree of mercurialization. Small doses of bromide will often produce much relief, and diminish the irritability and tendency to vomit—an important point, since many cases succumb to exhaustion when the disease appears to be subsiding. When the symptoms persist, and especially if the anterior fontanelle be tense, the advisability of an operation to drain away the fluid must be considered. Lumbar puncture is not appropriate, because the effusion is limited by adhesions which are probably not lower than the upper cervical region. The region of the fourth ventricle and the posterior arachnoid space can be reached by trephining the occipital bone close to the foramen magnum.[1]

Chronic hydrocephalus is the term applied to conditions in which there is an undue accumulation of fluid within the cranium, either in the ventricles (internal hydrocephalus) or between the dura mater and the arachnoid (external hydrocephalus). It is extended sometimes to include the œdematous condition produced by Bright's disease or anæmia.

Chronic external hydrocephalus is an extremely rare condition. It

[1] The reader desiring further information should consult the discussion upon Dr. W. Carr's paper read before the Royal Medical and Chirurgical Society (*Proc. R. Med. Chi. Soc.*, April 13 and 27, 1897).

is due to a chronic membranous inflammation of the dura mater and arachnoid, with effusion into the sub-dural space. It is complicated frequently by hæmorrhage into the false membrane. The effusion may be general, so that the brain lies at the bottom of the cavity. On the other hand, it may be limited by adhesion, so that in reality the condition is one of cyst.

Chronic internal hydrocephalus is the common form of "water on the brain." The effusion may be so great that the bones of the skull are forced apart and thinned, while the brain itself is a mere sack of nervous tissue enclosing the fluid. The distension may occur during intra-uterine life, the enlarged head may be the cause of difficult labor, and the child may die during parturition. Or the enlargement may first become noticeable from three to six months after birth, or in rare cases later.

The fluid in the ventricles has the normal chemical characteristics of cerebro-spinal fluid, and not those of dropsical effusion.[1] The accumulation, therefore, must be due either to an excessive secretion or diminished removal of the normal cerebro-spinal fluid. This fluid is present normally in the cerebro-spinal cavity (cerebral ventricles and central canal of the cord) and in the sub-arachnoid and sub-dural cavities. The fluid within and without the cerebro-spinal cavity is in communication through the foramen of Magendie, the aperture in the fold of pia mater which forms part of the roof of the fourth ventricle. This aperture may easily become obstructed by meningitis in the neighborhood. The obstruction may be in the iter, or at the foramen of Monro, in which case the dilatation is limited to the lateral ventricles. The choroid plexus may be found thickened and sclerosed, or the ependyma of the ventricles thickened and granular as though from antecedent inflammation. It is probable that in some cases the process is syphilitic. In acquired hydrocephalus the conditions may be similar, but the distension seldom attains the same degree. Among the causes must be mentioned posterior basal meningitis (*q. v.*), and tumours so situated as to obstruct the return of venous blood from the ventricles.

The quantity of fluid, especially in congenital hydrocephalus, may be very large, causing immense distension of ventricles, flattening and spreading out of the convolutions, and a distortion of the cranial outline, due in part to its great size, and in part to the separation of the cranial bones at the sutures, the frontal bone being tilted forward, the parietal bones outward, and the occipital backward. The general form of the cranium is globular when the enlargement begins in the

[1] Halliburton, "Chem. Phys. and Path." London: 1891, p. 358. The cerebro-spinal fluid should be classified with secretions rather than with transudations. "It is normally present in sufficient quantity to exercise a considerable amount of pressure."

first few months of life. When it begins after six months the increase in breadth is more marked, and the head has a pear shape, flattened above, where the anterior fontanelle, greatly enlarged, forms a large flat or slightly bulging area. In any case, the face, which is often emaciated, appears very diminutive in contrast with the distended skull (Fig. 68). The scalp is thin, and the hair of the head scanty. Owing to flattening of the orbital plates of the frontal bone the eyeballs are depressed, and the sclerotic is visible below

FIG. 67.

FIG. 68.

Fig. 67, "Mongolian" Idiot. To be contrasted with cretins Figs. 25 and 24 (Dr. Telford-Smith's case). Fig. 68, Hydrocephalus, chronic stage. The head was so heavy that it had to be propped against the back of the seat; the child having looked up with the head a little bent forward, the sclerotic is not well seen above the cornea.

the upper lid, while the iris may be partially covered by the lower. Congenital hydrocephalus occurs often in infants who present also spina bifida, cleft palate, or other faults of development.

Course.—Infants who suffer from congenital hydrocephalus, in whom enlargement of the head is present at birth or becomes conspicuous soon after, are weakly and ill nourished. They are dull, torpid, drowsy. They may suffer from convulsions, or from febrile attacks, after which they are somnolent or comatose. They seldom

survive many months, death being due to progressive exhaustion, or to an intercurrent disease (*e. g.*, broncho-pneumonia). If the enlargement begin after three or four months of age, it may be preceded by symptoms of meningitis, by convulsions, or merely by drowsiness and torpidity. The anterior fontanelle becomes tense and enlarges, the sutures separate, and the head assumes the shape already described. In the majority of cases the symptoms are progressive, and death ensues in a few weeks or months ; in others there are distinct remissions ; in a few complete arrest. Should this occur, ossification of the cranium proceeds, and Wormian bones often form, the skull is thin and long, remains pliable, especially at the anterior fontanelle.

Nervous **symptoms** due to chronic hydrocephalus are not definite. The child is dull in intellect, often idiotic. There may be divergent squint, and in severe cases blindness due to optic nerve atrophy. The limbs are weak, the lower often contracted. The **prognosis** is bad, since it is rare for infants who have once presented marked distension to survive beyond two or three years. If they do, they are stunted in mind and body as a rule, though in slight cases, in which arrest occurs early, intellectual development may not be very conspicuously incomplete. The **diagnosis** is easy once distension has been produced, although the mistake of attributing rickety enlargement of the head to hydrocephalus is sometimes made. The form of the head, the condition of the bones, and the concomitant symptoms ought to prevent such an error if ordinary care be taken.

The conditions upon which chronic hydrocephalus depends afford little scope for **treatment.** If a case of acquired hydrocephalus be seen in the early stage while enlargement is moderate but progressive, a mercurial course followed by potassium iodide should be tried. Whether the hypothesis that some of the more chronic cases are due to syphilis be correct or not, it is certain that a remission and, in a few cases, a complete arrest occurs under the use of mercury and iodide. This is the only treatment to which I have ever been able to attribute the least effect. The application of iodoform ointment to the scalp is useless, and so also is strapping the skull. The withdrawal of fluid from the ventricles by puncture made at the vertex a little to the right or left of the middle line, so as to avoid the longitudinal sinus, is, with antiseptic precautions, a justifiable operation, but as a rule the fluid accumulates again with great rapidity. Puncture followed by the injection of solutions of iodine or perchloride of mercury has not been followed by improvement. Diuretics and purgatives produce little or no permanent effect, although a single dose of calomel at the onset of one of the exacerbations will sometimes give considerable relief.

CHAPTER XLII.

INTRACRANIAL ABSCESS, THROMBOSIS, AND TUMOUR.

Abscess of the Brain: Course; Diagnosis; Treatment—Thrombosis of Cerebral Sinuses—Intracranial tumour.

Abscess of the brain.—Suppuration of the brain is, as a rule, confined to the white substance. It is very rare under one year and not common under ten years of age.[1] Abscess of the brain may be secondary to disease or injury of the cranial bones (including ear disease), or may be associated with suppuration elsewhere. In the latter case it is generally multiple, in the former frequently, the process being either a part of a general pyæmia or of pyæmic nature. By far the most common cause of brain abscess, especially in children, is disease of the ear. According to Körner's statistics, in 77 cases of brain abscess at all ages 25 were secondary to ear disease. The otitis may be recent and acute, or there may have been otorrhœa for many years. Among the rarer causes mention may be made of disease of the nose.

Abscess from *ear disease* in the large majority of cases is single, and is situated on the same side as the diseased ear, either in the temporo-sphenoidal lobe, or more rarely in one of the lobes of the cerebellum. In most cases—38 out of 40 (Körner)—the bone itself is diseased. Abscess of the brain, as well as in meningitis and sinus-thrombosis, secondary to otitis, begin, as a rule, at a point corresponding to that at which the inner surface of the bone is attacked. The roof of the tympanum enters into the middle fossa, and the bony partition is sometimes as thin as writing-paper; it is for this reason that disease of the middle ear most often causes abscess in the temporo-sphenoidal lobe which lies on the fossa. The mastoid cells are separated from the posterior fossa by a thin layer of bone, and hence abscess, secondary to disease in that region, is often situated in the cerebellum. The extension of the disease to the brain is due to thrombosis extending from the diseased bone, or from the ear, through the veins which pierce the roof of the tympanum; only rarely is there a direct communication by a suppurating tract. In

[1] Out of 223 cases Gowers found 24 from one to nine years, and 48 from ten to nineteen.

common with other forms of intracranial inflammation due to ear disease, abscess occurs more often on the right than on the left side.

The course of abscess may be acute or chronic, or rather intermittent. The mode of *onset* varies. There may be well-marked initial symptoms, resembling those of meningitis, which indeed is often present. The most prominent are headache and vomiting accompanied by pyrexia, which may be attended by rigors or general convulsions. This condition may run on into the terminal stage, or it may be succeeded by a period of latency. On the other hand, the earlier symptoms may all be those of this *latent stage*. They are headache, more or less constant and severe, accompanied by occasional nausea or vomiting. The attacks of headache may alternate with otorrhœa, the pain in the head coming on when the discharge stops. Convulsions, which may be one-sided or general, resembling idiopathic epilepsy and liable to be mistaken for it, are in some instances the first indications of cerebral disturbance. This stage may terminate suddenly by rupture of the abscess, usually into the meninges, producing acute general meningitis. The *terminal stage* is characterized by convulsions, pyrexia, and delirium followed by stupor. The onset of this stage in abscess due to ear disease is often determined by exposure to cold, by a blow on the head, or by the entry of water into the ear during bathing. Discharge from the ear is arrested, and unless a history can be obtained, the previous existence of ear disease may pass unsuspected.

The severity of the *headache* attending abscess varies; in abscess from ear disease it is often referred to the ear. It is intermittent or subject to exacerbations, as is shown in the young child by sudden fits of screaming, during which the child covers the ear with the hand or tears at it. Vomiting and giddiness are most prominent in cerebellar abscess, but may be present in cerebral suppuration. Grinding of the teeth and chewing motions of the jaws are often observed. Convulsions are as a rule general, but the onset of hemiplegia may be preceded by a one-sided convulsion. In some cases paralysis is replaced by well-marked rigidity affecting generally both lower limbs. Optic neuritis is observed more often in cerebral than in cerebellar abscess. The headache may be accompanied by photophobia, but ocular paralyses are on the whole rare, though ptosis is not very uncommon. In ear disease paralysis of the facial may be produced by the bone disease, but with these exceptions the cranial nerves usually escape. During the quiescent stage there is often mental depression, which may persist in the acute stage, passing into stupor and coma with little or no intervening delirium. The acute phases are accompanied by pyrexia, by sweating, and often by rigors, but in the quiescent stage there may be no elevation of temperature. Anorexia is a prominent symptom even in the latent

stage, and constipation is common. In the acute stage all nourishment is refused or taken with difficulty, the tongue is dry and brown and constipation obstinate. Both pulse and respiration are quickened in association with pyrexia, and may be irregular; towards the end the pulse may become very slow, and this may be so even when other symptoms are not well marked. Owing to the frequency with which cerebral abscess occurs in the temporo-sphenoidal lobe, localizing symptoms are commonly absent, but if the suppuration be immediately beneath the motor areas—an event most likely to occur in traumatic abscess—unilateral convulsions and paralysis may be produced. The abscess may rupture into the ventricles, producing general convulsions and coma, quickly followed by death. In cerebellar abscess the headache is occipital or is described as darting backward from the ear. Well-marked retraction of the head, due to meningitis of the posterior fossa, may be present and the abscess may rupture into this fossa, producing general convulsions followed by the symptoms of acute basal meningitis. The prognosis is extremely grave once the acute or terminal symptoms of abscess begin to develop. Unless relieved by surgical means, they terminate in death as a rule in four or five days or less, though life may be prolonged for a week or more.

The **diagnosis** of abscess within the cranium is often difficult owing to the resemblance of the symptoms, if acute, to meningitis, or if chronic, to tumor. A long history of intracranial symptoms, and especially the presence of cranial nerve paralysis and well-marked optic neuritis, is in favor of tumour, as is also retrogression of the symptoms. Even if evidence of meningitis exist, it must be remembered that it may be secondary to tumour. Pyrexia, especially if remittent, would point to abscess, but the main point in the diagnosis of this condition must be the recognition of some condition likely to determine it. The diagnosis from otitis is difficult, but pronounced cerebral symptoms and optic neuritis complicating ear disease, especially if accompanied by cessation of discharge, would justify a diagnosis of abscess (see "Sinus Thrombosis"). In young children ear disease may be suspected if, in the absence of evidence of local disease elsewhere, there be persistent heaviness, restlessness, constant whining and attacks of screaming, during which the head is bored into the pillow or rubbed against the nurse's arm, or if the child during the screaming carries the hand to the side of the head and tears at the ear.

The serious prognosis of cerebral abscess renders its **prophylaxis** a matter of great importance. The possibility of its development should be present to the mind in dealing with all cases of ear disease in childhood. It is most important to treat otorrhœa systematically, and to warn parents of the dangers of blows on the side of the head,

of exposure to cold, and of sea bathing, unless special care be taken to prevent the entrance of water into the ear. At the same time, every available means for the improvement of the general health should be taken, including the use of tonics and cod-liver oil. Residence in a dry, bracing climate is also to be recommended. The **treatment** of abscess so soon as a diagnosis can be made with reasonable probability is a surgical question. When there is reason to fear that abscess is developing, the child should be kept at rest in a cool, shaded room, an ice-cap applied to the head and warmth to the feet. It will generally be desirable to give a brisk purge (calomel and jalapine), and the diet should be very light. The value of morphia may be doubted ; it may possibly exercise a beneficial action on the progress of the inflammation, but by relieving the pain it may mask the symptoms and thus lead to the loss of valuable time.

Thrombosis of cerebral sinuses.—Thrombosis of a cerebral sinus in children may be primary, that is, due to a general marasmus, or may be secondary to a local disease, or injury of the bone or ear.

Marasmic thrombosis is very rare, or, at least, rarely diagnosed. The subjects are infants or children who have been brought to a condition of collapse by diarrhœa. For practical purposes, it may be regarded as occurring only in the superior longitudinal sinus. The symptoms are somnolence, apathy, vomiting and general convulsions. Cerebral anæmia (see "Spurious Hydrocephalus"), which is also brought about by diarrhœa in marasmic children, produces similar symptoms, so that the diagnosis of thrombosis can be made only when other special symptoms due to the impediment to the circulation are superadded. These are œdema of the scalp, of the side of the head, and of the forehead, epistaxis, and prominence of the fontanelle which has previously been collapsed. When the clot extends into the internal jugular, the external jugular will be over-full, and the thrombosed vein may be felt as a hard band.

Secondary thrombosis, in most cases, affects the lateral sinus, and is due to suppurative otitis media.

Körner[1] found the relative frequency of the forms of fatal intercranial inflammation to be as follows : sinus-thrombosis and pyæmia, 41 cases ; abscess of brain, 43 cases ; meningitis, 31 cases. Two or more of these conditions might be present together, so that there were altogether among the 115 cases, 50 examples of abscess and 53 of sinus disease. Körner states that more than half the cases of sinus disease (82 out of 151) occur in the first two decades, but the complication is much more common between ten and twenty (56 cases) than under ten (26 cases).

The thrombosis may be brought about by direct extension from

[1] *Die otitisch. Erkrank. des Hirns*, etc., 1896.

the inflamed bone, or by extension by accretion of a septic clot from the veins of the mastoid cells, which open into the lateral sinus. The clot may break down and produce secondary pyæmic abscesses, especially in the lungs. The **symptoms**[1] are the sudden onset in a person who has suffered for a year or more from purulent discharge from the ear, of fever accompanied by headache, vomiting, and pain in the affected ear, the discharge from which has ceased, as a rule. The fever is at first high, 103°–105° F.; the temperature soon falls to 100° F., or lower, but its course is very irregular. There is local tenderness and œdema over the mastoid process, and below the external occipital protuberance, and stiffness of the muscles of the back or side of the neck; in some cases optic neuritis develops eventually. The patient becomes apathetic, somnolent, or delirious, and, finally, comatose. All these symptoms are seldom present together, and it is justifiable to make the **diagnosis**,[2] if in the course of chronic suppurative ear catarrh there is a sudden cessation of catarrh accompanied by persistent pain in and around the ear, a high temperature, with marked fluctuations and frequent rigors, vomiting, rapid pulse, and constant headache. The occurrence of optic neuritis would clinch the diagnosis, but, in the presence of severe general symptoms, it will not be prudent to wait for its development before advising operation.

The **prognosis** is grave, though occasionally a sudden attack with headache, earache, vomiting, drowsiness, and fever may clear off after the spontaneous occurrence of a free purulent discharge from the ear.

The only effectual **treatment** is to give exit to the pus by operation, and it is essential that this should not be too long deferred.

Thrombosis may be secondary also to suppuration in the nose or eye, of the skull and scalp, or to erysipelas; thus, thrombosis of the *cavernous sinus*, a rare occurrence, may be due to extension from the ophthalmic veins (in phlegmonous inflammation within the orbit) or from the lateral or petrosal sinuses. The special symptoms it produces are a pushing forward of the eye, ptosis and paralysis of the sixth and other ocular nerves, and œdema of the lids and of the root of the nose.

Thrombosis of the *veins of Galen*, leading to effusion into the ventricles, has occurred as a fatal complication of scarlet fever.

Intracranial tumour is, relatively to the number living at the ages, rarer in childhood than in middle life. The actual number of cases of intracranial tumour in children is, however, large. Over 18 per cent. of the cases at all ages occur in the first decade of life, and 14 per cent. in the second.[3] The large number of cases in childhood is due, in the main, to the relatively great frequency of tuberculous

[1] Ballance, *Lancet*, vol. i., p. 1114, 1890.
[2] Milligan, *Lancet*, vol. i., p. 981, 1895.
[3] Gowers' "Manual of Diseases of the Nervous System," vol. ii., p. 454, 1888.

tumours. An analysis of Bernhardt's[1] statistics shows that of 59 cases under ten years of age, in which the nature of the tumour was stated, it was tuberculous in 37 (63 per cent.). In the second decade, of 45 cases, 13 were tuberculous. Next in frequency, are gliomata and sarcomata, then cystic parasites (hydatids and cysticercus). Gummatous tumours have been met with in childhood, but are very rare, as are also dermoid cysts and carcinoma.

Tuberculous tumours vary in size from that of a filbert to a walnut, or even larger. They occur most often in the substance of the brain, but occasionally spring from the membranes or from the surface of the brain. They are cheesy in the centre, but are surrounded by a grey zone of proliferation where the tuberculous process is spreading. In children under ten, several tuberculous tumours are found rather more often than a solitary tumour. The most usual site is in relation with the cerebellum; for the rest, they occur with about equal frequency in relation with the cerebral hemispheres, the basal ganglia, and the pons. In most cases, tubercle is found elsewhere in the body, and some cases terminate by tuberculous meningitis.

Gliomata are infiltrating growths; thus in the pons they may cause a uniform and symmetrical enlargement.[2] They vary in consistency, but are often soft, so that hæmorrhage is very apt to occur into them. They are found more often in children than in adults, whereas sarcomata, which spring often from the membranes and are more apt to perforate the skull, occur more often in adults.

The **symptoms** of intracranial tumour in children do not in any respect differ from those produced by like lesions in adults, except perhaps that owing to the more yielding state of the cranial sutures, and the greater frequency of tumours in the posterior fossa, obvious hydrocephalus is more often met with. The localizing symptoms are identical at all ages. The general symptoms are perhaps less easily recognized—headache, for instance, may be easily overlooked in a young child, especially if there be much somnolence. Optic neuritis may only develop at a late stage, and it is often very difficult to make a satisfactory examination. Vomiting is a frequent and, if carefully studied, a characteristic symptom; it comes on suddenly, usually without any sense of nausea, and is not influenced by the usual remedies. Sometimes after lasting for a day or more it ceases spontaneously, to recur again after some days or weeks. Somnolence—that is to say, a tendency to drop into a heavy sleep at odd times, or to sleep heavily at night, and to be dull and heavy by day—is a common symptom in children and may serve to excite suspicion. Convulsions occur with great readiness in children, and are usually general, or quickly become so even when at first they

[1] Bernhardt, "Beit. z. Symp. u. Diag. d. Hirngeschwulste," Berlin, 1881.
[2] *Vide* Money, *Med. Chi. Trans.*, vol. lxvi., p. 283.

present the true Jacksonian limitation. In tumour of the cerebellum, or so situated as to bring pressure upon it, giddiness is usually a marked symptom, and the gait is peculiar—clumsy and festinating—owing apparently to weakness or want of coördination of the muscles of the spine, and to some rigidity of the lower extremities. Internal strabismus from paralysis of the sixth nerve and enlargement of the head due to hydrocephalus are early symptoms in many cases of cerebellar tumour, as is also optic neuritis, which at a comparatively early stage may be accompanied by total loss of vision. General convulsions may occur, but in some cases, especially those in which the middle lobe is involved, there are attacks of tonic spasm with marked retraction of the head, and sometimes arching of the back. In such cases the retraction may eventually become permanent, and may be accompanied by marked rigidity of the muscles of the extremities.

The **prognosis** of intracranial tumour in children is worse than in adults, owing to the frequency with which the growth is tuberculous, and the rarity of gumma.

In **diagnosis** the main difficulty is to exclude functional disease. General convulsions may be epileptic or eclamptic, and unless optic neuritis or definite localizing symptoms develop, it may be impossible to distinguish the fits of idiopathic epilepsy from those produced by intracranial tumour even situated in relation with the cortex, for some paresis may remain after an epileptic fit. The mistake of attributing the early symptoms of tumour to hysteria is sometimes made; hysteria especially in girls about the age of puberty, is sometimes complicated by complaint of headache, said to be severe, and by retching or vomiting. The headache is, however, less severe, and the vomiting less sudden and uncontrollable, and other hysterical symptoms will probably be discoverable. The difficulty of diagnosis from intracranial abscess has already been mentioned (p. 475). In tumour the symptoms are more persistent, and develop more steadily and more slowly, paralysis of cranial nerves is more often present, the focal symptoms are more defined, headache is more constant, and fever is usually absent. The symptoms of tumour may resemble closely those of tuberculous meningitis if it run a chronic course, but are usually less constant, less well-defined, and the characteristic stages are not to be distinguished. If a tumour in relation with the cerebellum become complicated by meningitis the symptoms produced are those of posterior basal meningitis, and it must be borne in mind that chronic hydrocephalus may be produced by tumour cerebri.

The **treatment** of intracranial tumour is most unsatisfactory. Iodide of potassium produces amelioration or temporary recession of the symptoms, even in some cases which are not syphilitic. If the

growth is believed to be tuberculous the ordinary treatment for this infection may be advised with a certain amount of hope, as there is reason to believe that tuberculous growths within the cranium sometimes remain quiescent for long periods, and even undergo obolescence. If the symptoms point to the cortex as the seat of the tumour, the question of operation should be considered. For the relief of headache bromides are of service, as is also Indian hemp, but when very severe it may become necessary to give morphia or opium. Drugs exercise little or no influence over the vomiting, which is best controlled by rest in bed in a darkened room, and the administration of iced drinks.

CHAPTER XLIII.

HEMIPLEGIA. SPASTIC RIGIDITY. HEREDITARY ATAXY.

Secondary Hemiplegia—Congenital and Infantile Hemiplegia—Spastic Rigidity—Hereditary Ataxy.

Hemiplegia in childhood, excluding cases due to tumour, abscess, or acute meningitis (*q. v.*), may be (1) *secondary*—that is, it occurs as a complication of an acute specific disease, of heart disease, or is produced by an injury; or (2) it may be *congenital* or *acquired* in early life (*infantile*), when it involves an arrest of development.

Secondary hemiplegia which occurs as a complication or sequela of many acute diseases, including pneumonia, may be transient or permanent, complete or incomplete. Hemiplegia in which the paralysis is well marked and permanent must be due to coarse cerebral lesions, and such lesions have been found in many cases after death. They are due either to embolism secondary to endocarditis or to cardiac dilatation, or to hæmorrhage produced by the disturbance of intracranial circulation secondary to thrombosis of a cerebral sinus. Hemiplegia from these causes is met with in connection with scarlet fever, measles, diphtheria, typhoid fever, and small-pox. Lasting hemiplegia, and more limited paralysis coming on in whooping-cough during one of the paroxysms, is due to hæmorrhage produced apparently by the extreme venous congestion caused by the great rise of venous pressure which must take place towards the end of a paroxysm. The pathology of those cases in which the paralysis is transient is in need of elucidation. The hemiplegia of whooping-cough is in some cases exceedingly transient, and has been thought to be due to œdema, but in other cases it is permanent, or at least long lasting, and must be attributed to a gross hæmorrhage. On the whole it may be said that the prognosis of hemiplegia in association with the acute specific diseases, if the paralysis be not complete, is good. Great improvement and, in cases in which the extent and degree of paralysis has been slight, complete recovery is frequent, if not indeed the rule. On the other hand, the prognosis of well-marked hemiplegia in which signs of early improvement are absent is bad, more especially if there be commencing contracture, with ankle clonus and exaggeration of the deep reflexes.

In some cases improvement is rapid, and complete, or almost complete, recovery takes place. In others, the leg recovers almost completely, but the arm is permanently affected. In others, the leg recovers to a considerable extent, but there is some rigidity, and the patient has a hemiplegic gait. In others, again, there is well-marked permanent hemiplegia with rigidity. If the facial muscles are seriously affected there is usually imperfect growth of that side of the face, and often cranial asymmetry. In those parts in which palsy persists late rigidity ensues, and the deep reflexes are exaggerated. The elbow and wrist are more or less flexed, the fingers flexed or extended and rigid, all movements being slow, incomplete, and usually tremulous. The movements of the shoulder are generally more free, so that the hand can be placed on the head, and in some cases in which this cannot be done the failure is due rather to weakness than to rigidity. Only in a few cases is there permanent palsy without rigidity or increased reflexes.

With regard to the **treatment** of hemiplegia in childhood, it must be admitted that a good deal of scepticism is allowable as to the effect of the various means—massage, electricity, iodide of potassium, strychnine—which have been praised. Very remarkable improvement may be witnessed under any form of treatment in some cases. This occurs, as a rule, in those in which the paralysis is not complete in any part. In other cases presenting definite paralysis no treatment is of much avail. Massage is of use in maintaining nutrition and preventing deformity. In some cases the use of suitable prosthetic apparatus, preceded where necessary by tenotomy, will improve the power of walking.

At the time of onset the treatment must be such as is appropriate to the condition with which the hemiplegia is associated.

Congenital hemiplegia may be due to a lesion of one hemisphere occurring during intra-uterine life, involving an arrest of development, or to injury at birth—in almost all cases meningeal hæmorrhage (*q. v.*). If the lesion occur during intra-uterine life, the infant may be born with well-marked contracture of the affected side. If the lesion has been produced at birth there may be no symptoms to attract attention for some days, weeks, or months, when it is observed that the infant does not move the limbs on one side. In a minority of the cases convulsions, general or one-sided, and with or without retraction of the head, occur during the first few weeks of life. Even so the palsy may not be observed until some weeks later. The occurrence of epileptiform convulsions in later stages of congenital hemiplegia is considered under Epilepsy (*q. v.*).

Acquired infantile hemiplegia which comes on before or soon after the age of two years is occasionally a complication of an acute specific disease, but as a rule there is no discoverable determining cause.

The child is siezed with convulsions, one-sided or general, and suffers some elevation of temperature. The convulsions last for a few hours, or are repeated with few, and perhaps imperfect, remissions for several days. When they have passed away the child is found to be hemiplegic. In some cases, in which a series of fits occur, the palsy is at first slight, but becomes more complete after each succeeding fit. There may be no initial convulsions, but the child is suddenly found to be paralyzed on one side. The **pathology** of cases of this nature is probably not in all cases the same. Syphilitic arteritis, with consequent thrombosis, softening, and capillary hæmorrhages, may account for some; a localized meningitis, or meningo-encephalitis, for others. Strümpell's suggestion that in some cases the primary lesion is an acute encephalitis has received a certain amount of support from pathological observation,[1] and accounts probably for many of the worst cases. In infantile hemiplegia, whether produced by injury at birth, or by disease, the **permanent symptoms** are due to cortical sclerosis and atrophy. The sclerosis may be (1) widespread, involving the whole of one hemisphere; (2) limited to the motor area; (3) scattered, the great overgrowth of fibroid tissue producing nodular projections; or (4) associated with cysts at the surface, or porencephalus—cavities extending into the substance of the brain, and even reaching the ventricle.

In a minority of cases the paralysis, especially if from the first it have been incomplete and not very extensive, gradually clears away. More often considerable improvement occurs in the lower limb, so that the patient learns to walk, but has a hemiplegic gait more or less marked. In almost all cases the paralysis is greater in the upper limb. The arm is carried usually in contact with the side, the forearm flexed at the elbow is carried across the trunk, the wrist is flexed and the fingers are adducted and flexed at the metacarpo-phalangeal and inter-phalangeal joints. As a rule late rigidity ensues, and is the most marked symptom of the condition. In such cases the deep reflexes are exaggerated.

The mental condition varies a good deal. Some of the children thus afflicted appear to be of average intelligence, more often they are dull and slow, some are aphasic, not a few obviously imbecile. In other instances epileptic seizures occur, and the mental state deteriorates rapidly. In a considerable proportion of cases the affected limbs become subject to involuntary movements, tremors, choreiform movements (post-hemiplegic chorea), or athetosis (see below). There is a possibility that hemiplegia coming on in infancy or early childhood may be due to syphilis, and it is always justifiable to give a mercurial course followed by the administration of iodides;

[1] Reymond, *Jahrb. f. Kinderhlkde.*, Bd. xliv., S. 157.

for even if the lesion be not syphilitic, this line of treatment may have a beneficial effect on the inflammatory process which it must be assumed is present in, at least, a large proportion of the cases. Massage is of use in improving the nutrition of the muscles. In certain cases the powers of walking may be greatly improved by suitable prosthetic apparatus.

Spastic rigidity.—Under this general head may be conveniently classed together cases of nervous disorder, usually congenital, characterized by rigid spasm of the lower, sometimes of all four extremities, and occasionally of the neck and trunk also.

The pathology is not the same in all cases, though in all there is an atrophic condition of an area, more or less wide, of the cortex. In some, and these are the majority, the condition is truly congenital and is due to meningeal hæmorrhage (*q. v.*) occurring during the act of parturition. In others it is due to an imperfect development of the pyramidal tract. In a child born at term this tract is not yet completely developed, and it seems reasonable to suppose that any undue pressure during delivery would be liable to damage this tract in particular and so hinder its subsequent normal development, more especially if, as has been the case in a good many instances, the child has been born prematurely. The same woman may bear more than one child which suffers from spastic rigidity, though not always to the same degree or with the same distribution. In other cases the condition develops after an acute illness in early childhood. Such cases doubtless belong to the same category as those considered under the head of acquired hemiplegia. There is also an hereditary form which may begin later in life, even in middle age, due to degeneration of the pyramidal tracts. The association of spastic rigidity with mental deficiency, nystagmus, and atrophy of the optic nerve is due to a cerebral lesion, to which probably the spinal degeneration is secondary. Mental deficiency is, however, common in children with spastic rigidity who present no other evidence of cerebral lesion.

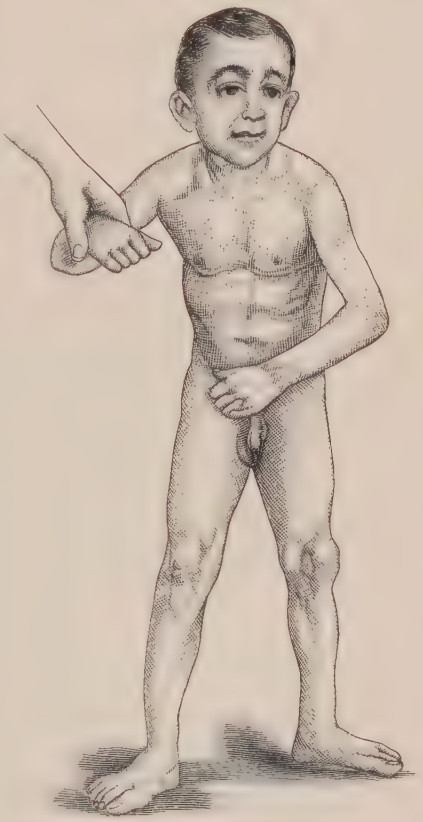
Massolongo[1] distinguishes five main clinical types, but contends that the difference in extent and character of the symptoms is due less to the involvement of different nervous areas than to the varying period of life at which the primary lesion is produced. His five types are:—(1) General spastic rigidity; (2) paraplegic rigidity; (3) bilateral spastic hemiplegia; (4) bilateral athetosis; (5) congenital spastic chorea.

In general spastic paraplegia, the form most commonly seen, advice is sought, usually, when the child is between one and two years old, because its legs are stiff, and it does not learn to walk. It has been born prematurely, and usually in a state of asphyxia, due sometimes, but not always, to prolonged labor, from which it was with

[1] *Il Policlinico*, vol. iv., m., fasc. 1, 2.

difficulty recovered. The muscles of the lower extremities are in a state of rigid spasm. The thighs are rotated inwards, and brought into apposition by spasm of the adductors; the knees are in contact, but, owing to the inward rotation, the tibiæ are separated by a considerable interval, and the feet are in the position of equinus. When a little older, the child may attempt to walk when supported under the shoulders. The foot is brought forward by a semicircular movement, during which the trunk is bent towards the opposite side; but, in spite of this tilting of the pelvis, the toes are dragged along the ground. The adduction may be so extreme that the foot is brought to the ground in front of the other. Lastly, as the weight is transferred to the toes, the elongation of the gastrocnemius causes an immediate reflex contraction which throws the whole body forward; this gives to the gait, if the art of walking is ever acquired, a peculiar jumping, hurrying character. In mild cases, the upper extremities escape, but they may be affected by rigidity to any degree, as may also the muscles of the neck. Indeed, in severe cases, all the muscles of the trunk may be more or less involved, except those of respiration. When this is the case, the head may be retracted, while the trunk is bowed forward. Sitting may be impossible, owing to the rigidity of the hip and trunk muscles. If it be possible, the equilibrium is unstable—the weight rests on the ischial tuberosities, the thighs are semi-flexed on the trunk, the knees flexed to an obtuse angle, and the feet held rigidly forward. Strabismus, usually convergent, is present in about one-third of the cases, and in many instances is associated with errors of refraction. Owing apparently to some rigidity of the face muscles, the expression is stupid. Speech is generally drawling and jerky, and is acquired late. There may

Fig. 69.



General spastic rigidity; showing the attitude in standing. (After a drawing made by A. Dall' Occa Bianca for Massolongo.)

not be any marked intellectual defect, though the character is usually irritable, capricious, and often mischievous. There may be some difficulty in deglutition, but the sphincters are not affected as a rule, though, in a few cases, there is incontinence of urine. The deep reflexes are exaggerated, but when the rigidity is extreme, it may not be easy to elicit them; ankle clonus can be obtained in many cases. The superficial reflexes and common sensation are unaffected.

In some cases, the spastic conditions may be limited to the lower extremities (*paraplegic rigidity*), or is very much more marked than in the upper limbs. In other cases, again, the paraplegic rigidity is combined with spastic paralysis of one upper limb (congenital hemiplegia). In other cases, again, in which the condition is in its most pronounced form, there is *congenital diplegia* with spastic rigidity. It is in such cases that **athetosis** seems to be most liable to occur. In this condition, the limbs are subject to spontaneous involuntary slow movements. Thus in the upper limbs slow spasmodic movements of extension, abduction, and flexion of the fingers, of flexion and rotation of the wrist, of flexion and extension of the elbow, and of rotation, abduction, and adduction at the shoulder-joint, may be observed. Similar movements may affect the lower limbs. Occasionally the facial muscles are affected. The movements do not, as a rule, occur during sleep. They may be aggravated, or produced by emotion, but are not painful. The general character of athetotic movements is well illustrated in the following illustration (Fig. 70) of a case of Massolongo's. In other cases, the extremities are subject to almost constant slow spasmodic movements, resembling those of chorea, whence the term **congenital spastic chorea**, applied to this condition.

In spastic rigidity, the *mental* condition varies a good deal. When the lower limbs only are affected, intelligence may be fair; when the symptoms have a hemiplegic distribution, the acquisition of knowledge is generally much hindered, and the child appears dull in intellect. In the cases in which the lesion is more extensive, the patients are almost invariably quite imbecile.

The **prognosis** even in the mildest case is bad, inasmuch as complete attainment of muscular power cannot be hoped. In all but the most severe, some improvement generally takes place. The rigidity may, for example, disappear from the upper limbs. This result is favored by systematic active and passive exercise of the limbs, and by massage. Tenotomy may be necessary to obtain the full advantage of this treatment. In some cases, the aspect of the child, which suggests complete imbecility, may be, to some extent, misleading, and a good deal of knowledge may be imparted by a painstaking instructor.

Hereditary ataxy (*Friedreich's Disease*) is a form of ataxy which comes on in childhood or early life, and is due to degeneration of the posterior columns of the spinal cord.

It is a family disease, *i. e.*, a disease which attacks commonly several members of the same generation, brothers and sisters of one family, although isolated cases may be met with. The first symptoms come on usually at the same age in each member of the family attacked. At or shortly before puberty is the most common period for the onset, which appears sometimes to be determined by an attack of measles or scarlet fever or some other acute infectious disease, but beyond this nothing can be said as to etiology.

FIG. 70.

Bilateral athetosis (congenital). (After a drawing made by A. DALL' OCCA BIANCA for MASSOLONGO.)

In cases examined after death the cord has always been small and has shown widespread sclerosis—in the posterior columns (columns of Goll in their whole extent, and columns of Burdach in their upper part), in the direct cerebellar tract extending laterally into the column of Gowers, in the lateral columns (crossed pyramidal tract), in the grey matter (columns of Clarke, and posterior horns). In some cases dilatation of the central canal has been observed.

The most characteristic **symptoms** are those affecting the motor system. The patient stands with the feet far apart and has difficulty in maintaining his equilibrium; the body sways, and the feet are shifted to maintain the upright attitude; the unsteadiness may or

may not be aggravated by closing the eyes. The gait is reeling, the steps short and uncertain; on the whole it resembles more the gait of intoxication than of locomotor ataxy, in which there is much more incoördination. While walking or standing the head is nodded or moved unsteadily. In some cases there is a distinct tremor of the limbs and head, and choreiform movements of the same parts. Paresis and wasting of limb muscles may be present, and ocular paralyses have been observed. Sensory disturbances are rare; darting pain has been present in some cases, but not anæsthesia, analgesia, nor, it would seem, loss of muscular sense. The tendon reflexes are lost, or greatly diminished, the cutaneous unaffected. The movements of the pupil are not disturbed, and there is no affection of vision or optic atrophy, but nystagmus is observed in most cases, though it may be a late symptom. Vertigo, permanent or paroxysmal, is common, and the patient may suffer from severe headache. Development of the intellect is not retarded, but the child appears stupid owing to the speech being slow and hesitating, though some phrases are blurted out. The expression is often heavy. The genito-urinary system is not involved. Lateral curvature is common in the late stage. A rather characteristic deformity is a peculiar clubbing of the feet. The foot is short, the instep high and hollow, while the toes are over-extended. This retraction and over-extension of the great toe may be the first symptom of the disease to attract attention. In some cases loss of the knee-jerk may be observed earlier, but the condition of the tendon reflexes is rather uncertain, and their retention in the early stage would not negative the diagnosis of Friedreich's disease. The reeling gait is the next symptom to be observed, and the unsteadiness increases until the patient, after perhaps three or four years, becomes unable to stand, and is henceforth confined to a chair or bed. He succumbs, usually, to some intercurrent disease, the onset of which is favored by his inactive existence. Recovery is not known ever to have occurred, although the progress of the symptoms may show intermissions.

Treatment, whether by drugs, electricity, or massage, has not been shown to exercise any influence on the course of the disease.

Hereditary cerebellar ataxy has been described by Nonne and Marie. It appears to be due to imperfect development of the cerebellum and cord. The symptoms, which are first observed about puberty, differ from those of Friedreich's disease inasmuch as the deep reflexes are retained and become exaggerated, Paralysis of the pupil to light and in accommodation and diminution of the field of vision due to optic atrophy are present, and common sensation is disturbed. Lateral curvature and clubbed foot have not been observed.

CHAPTER XLIV.

LESIONS OF NERVES.

The Motor Nervous Apparatus—Reaction of Degeneration—Birth Palsies—Facial Paralysis—Multiple Neuritis.

The path of motor innervation from the cortex cerebri to the muscle consists of two distinct nervous structures (neurons) which are not in direct communication. The upper neuron is the cortical pyramidal cell with its dendritic processes in the cortex, and its axis cylinder consisting of a number of fibrils each with its separate destination. The axis cylinder fibrils descend in the pyramidal tract of the spinal cord and end in arborescent processes which interlace with the dendritic processes of the spinal multipolar cell, with which they thus come into relation, although there is no actual continuity of substance. The lower neuron consists of the spinal multipolar cell with its dendritic processes in the cord and its descending axis cylinder process, which ends in an arborescence by which it is brought into relation with the muscle fibre. The cell in either neuron may best be regarded as its centre of nutrition. Destruction or degeneration of the cell involves degeneration of its axis cylinder and the arborescence by which it terminates. Destruction of the pyramidal cell or interruption of the axis cylinder causes degeneration of the part below down to the arborescence in the cord, and consequently the transmission of voluntary impulses from cortex to cord is abolished. Similarly, degeneration of the multipolar cell in the cord involves degeneration of the axis cylinder in the cord and motor nerve, and of the terminal arborescence and the muscular end plate. When this degeneration of the nerve reaches the muscle it is attended by rapid atrophy of the muscle, or of those muscular fibres which are in relation with the axis cylinders of those multipolar cells which have been damaged. We have an example of damage of the upper (cerebral) neuron in ordinary hemiplegia, of damage to the lower (spinal) neuron in infantile paralysis. But through the nutrition of the muscle fibres is intimately bound up with that of the spinal neuron, yet muscle is a mesodermic structure and can develop independently of nervous impulses—a fact which explains the circumstance that muscular fibre is subject to certain errors of development and nutrition which are produced independently of lesion of

nerves. Of this event pseudo-hypertrophic muscular paralysis affords a well-known instance.

Destruction of the nucleus of a nerve in the case of the spinal motor nerves, of the multipolar cells of the anterior cornu, is followed immediately by loss of function; while severance, whether by injury or disease, of the organic connection of the nerve with the ganglion cells produces loss of function in the part below the lesion. The withdrawal of the nerve from the influence of the ganglion cells entails degeneration of the nerve tubules. The axis cylinder breaks up into smaller and smaller segments, until it finally disappears, the medullary sheath being destroyed in a similar manner. The process, which is complete in about a week, extends from the centre towards the periphery. If the damage to the nerve be slight, the degeneration does not affect all the fibres, and restoration of function then takes place more rapidly. It is a slower process than degeneration, but, like it, extends from the centre outwards. The axis cylinder is first restored, the sheath later, and conductivity may be reëstablished before this, and before the nerve can be excited electrically.

The degeneration of motor nerves is succeeded, in about two weeks, by degeneration of the muscle fibres; they shrink, their striation is blurred, and they become granular. If the nerve is not regenerated the striation is lost and the muscular substance gradually disappears, while there is at the same time a growth of connective tissue, which generally involves some diminution of the length of the muscle as well as of its girth. In lesions short of total division some fibres in a muscle escape, while others are totally destroyed.

Reaction of degeneration.—In health a faradic current of sufficient strength applied to the *nerve* produces a continuous contraction of the muscle; the galvanic, a momentary contraction when the current is made and broken only. When the nerve is diseased a stronger faradic or galvanic current is needed to produce contraction, until finally, when degeneration has taken place, no current which can be used produces any contraction. In health either current applied to the *muscle* produces contraction; the response both to the galvanic current and to the faradic is quick, being in both instances due to stimulation of the nerve-endings. With lesion of the nerve, and consequent degeneration of the nerve-endings, the faradic current produces no contraction, but since the galvanic current is capable also of stimulating the muscle fibres themselves, a contraction follows application, though more slowly than when the nerve-endings are healthy. After the degeneration has progressed to a certain stage, which is reached the earlier the more severe the case, this response of the muscle fibres to the galvanic current becomes more ready than in health. To this quantitative change is added a qualitative change. In health the weakest galvanic current which causes

contraction of the muscle does so when the current is made with the negative pole on the muscle (kathode closure contraction, K.C.C.). When the nervous mechanism has degenerated a contraction may occur with as weak or with a weaker current when the positive pole is on the muscle (anode closure contraction, A.C.C.), and contractions may occur also with the same current when it is broken (anode opening contraction, A.O.C., and kathode opening contraction, K.O.C.[1]). To this altered qualitative and quantitative reaction of nerve and muscle to the electric currents the term "reaction of degeneration" is applied. It is not always as definitely marked as is above described. When the damage to the nerve is slight the irritability of the nerve to both currents may be retained, and the only evidence of the existence of a reaction of degeneration is increased muscular irritability to the galvanic current, with some change also in the order of contraction to the poles (qualitative change). On the other hand, in very chronic changes the loss of irritability proceeds *pari passu* in nerve and muscle, and the reaction of degeneration is not to be observed.

With the regeneration of the nerve, recovery of function takes place, the rate of recovery depending mainly on the severity of the lesion. Voluntary power is first regained, then the galvanic reactions become normal, and, lastly, the faradic.

Anæsthesia, which is the eventual result of degeneration of a sensory nerve, may be preceded by a condition of hyperæsthesia. The anæsthesia is often incomplete, especially in the hands and face; in a mixed nerve, a lesion, capable of producing paralysis of motion, may be accompanied by little loss of sensation. Trophic changes seem seldom to occur in children as an accompaniment of lesions of sensory nerves.

Birth palsies.—Certain nerves are liable to injury during the act of birth. Thus the facial may be damaged by the direct pressure of one blade of the forceps, or may be compressed by extravasation into the parotid. The brachial plexus may be injured by traction made by the finger or blunt hook, or by compression of the shoulders in dystocia.

Facial paralysis may be due to damage to the conducting tract above the nucleus in the pons (see "Hemiplegia"), or to damage of the nucleus or trunk of the nerve. It is occasionally observed as part of diphtherial palsy. Meningitis of the base may involve the facial along with other cranial nerves. The two chief causes of facial paralysis in infants are injury during parturition and middle-ear disease. Paralysis from cold is very rare in young children, but is not very uncommon after the tenth year. Facial paralysis may also be secondary to adenitis, the trunk being compressed by the enlarged

[1] The normal order is: K.C.C., A.C.C., A.O.C., K.O.C.

glands, or perhaps involved in the inflammatory process, or at a later stage, distorted by cicatricial contraction after suppuration.

Facial paralysis, associated with extravasation into the parotid, is of short duration, and, as a rule, that due to pressure by the forceps recovers within two or three months; but in a few cases the injury has been severe enough to cause degeneration and permanent paralysis, with defective development of the face on the affected side. Congenital paralysis of the facial, not due to injury, has also been seen; it is permanent, but its pathology is unknown.

Lesion of the nucleus or trunk of the facial nerve is followed in a few hours, or, at most, a couple of days, by loss of tone and movement of all muscles of the face. The loss of tone is not so perceptible in a child as in an adult, owing to the greater plumpness and elasticity of the cutaneous structures, but the paralysis is evident when the child laughs or cries. In emaciated infants, the loss of tone is marked. When the attempt is made to close the eye, the eyeball is rolled up but the eyelids are not closed, nor are they closed in sleep. Except in marasmic infants, the lower eyelid does not fall away from the globe as in the adult, and for this reason the overflow of tears may not be very noticeable. Owing to the paralysis of the buccinator, curd and scraps of food are apt to accumulate between the jaws and the cheek, and so to cause stomatitis. Facial paralysis, associated with middle-ear disease, is of bad prognosis in infants. They are, as a rule, marasmic, many succumb to tuberculosis, and it seems not improbable that the ear disease may be tuberculous from an early stage, if not from the first. The prognosis of facial paralysis following exposure to cold or produced at birth is good; the degree of paralysis may not be the same in all parts when the patient comes under treatment, since recovery begins earlier in the upper than in the lower. Slight cases may recover completely in a fortnight, but the average duration is two or three months. When of longer duration, contracture of the muscles of the paralyzed side ensues, and by causing the naso-labial fold to develop prematurely, produces a deformity of the face which may last long.

The **treatment** of facial paralysis, due to middle-ear disease, must, in the first place, be directed to the cure of that condition. The only effectual treatment for the paralysis itself is by electricity. For the first week, the faradic current should be used to the muscles themselves; subsequently it may be supplemented by the galvanic current, but the use of the faradic current should be continued two or three times a week so long as the movements remain defective. Counter-irritation is probably quite useless.

Children are subject to the various forms of paralysis usually attributed to **multiple neuritis**. The lesion is produced by toxic bodies,

either such as are introduced into the body by accident (arsenic, lead) or design (arsenic, alcohol), or such as are produced during various diseases—for example, the acute specific diseases (*q. v.*). Though the main stress of the toxic influence falls on the nerves, there is practically no doubt that the whole neuron suffers, and that the cells of the spinal cord do not escape. The characteristic symptom is the association of motor with sensory paralysis. The paralysis is usually symmetrical, and affects all four extremities to a greater or less extent. It would appear that certain groups of muscles are more readily affected by these chronic forms of poisoning than others. The extensors of the wrist and of the foot are those most often attacked, so that wrist-drop and foot-drop are common symptoms. Pain in the area of distribution of the affected nerves, due to involvement of sensory fibres, is common; and in some cases the nerve trunks are swollen and a little enlarged. The persistence of such pain, and especially the detection of enlargement of the trunks, are important points in diagnosis. The deep reflexes are almost invariably diminished or lost, usually before the paralysis develops. All forms of sensation become diminished. The electrical reactions are very variable, but wasting of the muscles is usually an early symptom. The paralysis and wasting may be attended by contracture, and the production of various deformities, especially talipes equinus. When recovery has commenced sensation returns, as a rule, at a much earlier date than the recovery of muscular power.

The main point in **treatment** is to recognize and remove the cause. During the early stage, when pain is a prominent symptom, warm baths and warm applications to the part give relief. Later, when paralysis becomes evident, the galvanic current should be used without delay, and later still, gentle massage and regulated or rhythmical exercises are valuable.

CHAPTER XLV.

AMYOTROPHY.

Muscular Atrophy—Infantile Paralysis—Progressive Neural Muscular Atrophy—Primary Muscular Dystrophies—Pseudo-hypertrophic Muscular Paralysis.

Paralysis attended by or dependent on degeneration of muscular fibres may be due to lesion of the nervous mechanism which supplies the muscles, or to primary lesion of the muscles themselves. The first group has been further divided into those dependent on changes in the anterior cornu and those due to lesion of motor nerves. The number of varieties of chronic amyotrophic disorders described is very large. The distinctions recognized are in the main founded on the anatomical distribution of the paralysis. It is probable that the classification and relations of diseases of this group will shortly undergo modification, but for the present, however, it will be convenient to retain the classification into three types—

Primary lesion in cord	Anterior poliomyelitis. (*a*) Acute infantile paralysis. (*b*) Chronic progressive muscular atrophy (Aran-Duchenne).
Primary lesion in nerve.....	Progressive neural muscular atrophy (Charcot-Marie-Tooth).
Primary lesion in muscles (primary muscular dystrophies).....	Muscular pseudo-hypertrophy (pseudo-hypertrophic paralysis). Juvenile form of progressive muscular atrophy (Erb's).

Infantile paralysis is an infective disorder seldom or never encountered after childhood. The characteristic lesion is rapid degeneration of the motor cells of the anterior horns of the connected motor nerves and nerve-endings, and atrophy of the muscles supplied by these nerves.

The **etiology** of the disease is obscure. It has occurred occasionally in epidemics, and the symptoms at the time of onset—that is, at the time when in the most typical cases the damage to the nervous structures is produced—resemble those of an acute infectious disease, but infection from one case to another has never been traced. The lesion of the cord and nerves is due, probably, either (*a*) to a microbe

having a special affinity to the nervous system, or, (*b*) as seems more in agreement with all the circumstances, it is due to the poisonous action of some product of microbial activity elsewhere in the body, which produces its most marked structural effect on the delicate and growing nervous structures of the child, inducing definite degenerative changes. Paralysis having the same characters is an occasional complication of various acute specific diseases.

The distribution of the *lesion* in the *spinal cord* proves almost conclusively that the cause is vascular. It is an acute cellular degeneration (myelitis), limited in extent both in the vertical and in the horizontal planes. The focus of the myelitis is in the anterior cornu on one side, but it may extend slightly into the white matter of the antero-lateral column. Within the focus the large motor cells undergo granular degeneration, and either disappear altogether or become converted into rounded masses without processes. The nerve fibres also undergo granular degeneration. The blood-vessels are dilated, sometimes thrombosed, and proliferation occurs subsequently in the nuclei of their walls. Later, changes of cicatricial nature take place. In the affected region the diseased side of the cord is smaller than the healthy. This is due in part to the destruction of nervous elements and contraction of fibrous tissue in the anterior cornua and adjacent part of the antero-lateral column, and in part to associated defective development of other portions of the cord at the level affected. The extent of this area in the vertical direction varies from about a quarter of an inch to one inch. When more than one limb is affected, there may be two or more such areas of shrunken cord.

The symptoms of the disease fall into three stages :—Onset, regression, and the stationary stage, or stage of deformity.

The *onset* is attended as a rule by fever, gastro-intestinal disturbance, and nervous symptoms. There may be somnolence or coma, excitement with clonic spasms, or general convulsions. Death may occur at this stage owing to the respiratory centre being involved. In some cases there is considerable pain in the limb generally referred to the joints, but often there are no obvious localizing symptoms. Either during the febrile stage or, more commonly, as it subsides the child is found to be paralyzed more or less extensively. The paralysis may affect one lower or upper extremity, or both lower extremities, or one upper and one lower extremity on the same or opposite sides, or all four limbs. In other cases the onset, though sudden, is not attended by marked constitutional symptoms, the child being merely found, when taken up in the morning, to be paralyzed in one limb.

The stage of *regression* begins in a few days. The complete paralysis of a limb or limbs begins to clear up, and at the end of a week

or a fortnight some power has been regained in all the muscles of the limb except those of which the nervous connections have been permanently damaged. In a few instances complete spontaneous recovery takes place, but in the ordinary course certain muscles remain paralyzed, as is shown by the attitude of the limb and by the inability to perform certain movements. These muscles waste rapidly, and their electrical reactions are found to be altered. Faradic irritability is lost rapidly after the onset, and those muscles which are to be permanently paralyzed do not, a fortnight after, respond at all to the current. The affected muscles on the contrary react very readily to the continuous current, and present the reaction of degeneration. There is often a good deal of pain in the limb at this stage, and the joints may be hot and tender, but cutaneous sensation is unaffected.

The stage of *deformities* follows gradually upon that of regression. The wasting of the affected parts becomes very conspicuous, the limb is constantly cold, and its general nutrition suffers. It grows less rapidly than the sound limb, the bones are not only shorter but slighter, and perhaps more fragile. The skin is easily damaged, and is particularly liable to become the site of chilblains. Local œdema is easily induced by a dependent attitude frequently assumed, or by the pressure of garters, and cyanosis and mottling of the surface on the least exposure afford further evidence of defective circulation and nutrition. The amount of subcutaneous fat may be somewhat excessive ("subcutaneous adiposis," "local obesity"), but more often it is deficient.

The *distribution* of the paralysis is governed by functional, not by anatomical relations. Thus the supinator longus is affected along with the biceps, brachialis anticus, and deltoid ("the upper arm type" of Remak), while in the so-called "forearm type" the triceps is paralyzed, but the supinator longus escapes. In the "lower arm type" the extensors or flexors of the wrist and fingers are paralyzed. In the lower limb the peroneal group of muscles is that most frequently affected, then the posterior tibial, next the quadriceps in association with the tibialis anticus—muscles associated in the extension of the leg in walking. The glutei and the hamstrings are not often affected, the muscles of the face very rarely; the sphincters escape even at the height of the disease. Nearly all the muscles of a limb may be affected, so that the leg, for instance, is flail-like, and quite useless.

The **treatment** of infantile paralysis is usually regarded as a somewhat hopeless task, inasmuch as those muscles which are supplied from the parts of the cord where the lesion is most intense become more or less completely paralyzed and atrophied, while those supplied from parts less severely damaged recover spontaneously. It is reason-

able to assume, however, that the lesion in the cord is, in all parts which are at all affected, the same in kind, though it differs in degree; and further, that the axis cylinders and the end plates suffer at the same time. It is usually held that no treatment directed to stimulate the activity of the cord and nerves is permissible in the early stage. It may be argued, however, that as the nervous structures are extravascular the fear of exciting or maintaining inflammatory action in their neighborhood by stimuli, such as electricity, which influence only or chiefly the nervous structures, need not be entertained. This view has been energetically defended by Cagney,[1] who argued further that since the peripheral ending of the motor nerve besides participating in the degeneration of the spinal cell, is, in virtue of its own blood supply, still further involved in the toxæmia which is the assumed cause of the disease, it would follow that a peripheral neuritis is superadded to the changes in the cord, the part most affected in the toxæmia. If this be so, it is justifiable to endeavor to maintain the nutrition by stimulation applied to the periphery from a very early stage. Even if these views be not accepted to the full, it must, I think, be admitted that the fear of increasing the damage to the nervous elements has been made rather a bugbear. In whatever light we regard the primary lesion in the cord, it will not be denied that it reaches its maximum in a very short time, for the stage of febrile reaction is short and is, indeed, not always present, and that regression of the paralytic symptoms—that is to say, the recovery of the anterior cornua at those levels which have been primarily affected to a lesser degree—begins in a few days.

If the patient is seen during the febrile stage a dose of calomel should be given at once, and the same drug should be prescribed in small doses for some days. Belladonna and ergot both have their advocates, but it is doubtful whether they exert any influence on the course of the disease. The patient should be kept warm, and, if possible, in bed in the recumbent attitude. Cagney, in accordance with the principles indicated above, advocated the employment of weak galvanic currents, massage of the affected limbs, and injections of strychnine from the earliest stage. He maintained that the prospect of recovery depends upon the promptitude with which these measures are undertaken. He informed me that in some severe cases, in which under the expectant treatment a large remnant of permanent paralysis was to be looked for, complete recovery of function was eventually obtained. The affected muscles continue to display the galvanic reaction of degeneration for many months, and he

[1] His contention is stated briefly in a note of a paper contained in the *British Medical Journal*, 1896, vol. ii., p. 1506. The lamented death of Dr. Cagney occurred while these pages were going through the press, and he has left no further published record of his experience.

looked on the persistence of this phenomenon as a hopeful sign, and as an indication for the continuance of treatment. At the same time the patient should take fatty foods, cod-liver oil and extract of malt, and other foods and digestives which maintain nutrition. The affected limbs should be kept wrapped in cotton-wool. The amount of strychnine injected should be at first $\frac{1}{100}$ of a grain (of the nitrate). Cagney injected it into the substance of the affected muscles daily, and attached considerable importance to this. He increased the dose gradually to as much as $\frac{1}{10}$ or even $\frac{1}{5}$ of a grain. The affected muscles should be galvanized daily, using the current of from 10 to 20 cells; at the commencement of treatment the positive pole should be applied locally, afterwards the two poles alternately.

At a later stage massage is most useful, and should be continued throughout the whole period of growth. It is often said that after the expiration of one year from the onset no further improvement can be expected. Even if this be true, it is certain that deterioration may take place if the muscles are not used. This wasting and loss of power may undoubtedly be checked by massage well applied, and faradic electricity is also of some use for the same purpose. Massage further has the effect of improving the circulation through the limb generally, and thus tends to maintain growth and to prevent the shortening of the limb, which is often one of the main causes of extreme lameness.

The immediate effect of *massage* on the muscles manipulated is to cause an increased flow of blood through them.[1] When the massage is stopped there is a momentary accumulation of blood in the muscle substance, followed by a greatly increased flow. The effect of massage of a group of muscles is, in its influence on the local circulation, analogous to that of the contraction of the same muscles. The effects of massage on the general circulation of a considerable muscular area are to produce a lowering of peripheral resistance in the area, and as a consequence of this more blood is propelled at each heart beat from the arteries into the veins, with an attendant fall in arterial tension.

Prosthetic apparatus suitably adjusted may be of very considerable use in improving the power of walking and in checking the increase or production of deformities. It should be as light as possible in construction. Before it can be properly applied it may be necessary to perform tenotomy.

Progressive neural muscular atrophy, often spoken of as the peroneal form of muscular atrophy, begins as a rule in the lower extremities. The usual course of symptoms is that weakness followed by atrophy affects first the extensor muscles of the toes, then

[1] Lauder Brunton and Tunnicliffe, *Jour. of Phys.*, xvii., p. 365.

the small muscles of the foot, then other muscles of the lower extremities. The two legs are attacked almost simultaneously, or in rapid succession, and eventually talipes equinus or equino-varus is produced. The atrophy may involve the muscles of the upper extremities and may even begin in them, but the legs are always involved early and to a greater degree. Sensory changes also occur, especially hyperalgesia, but tactile sensation and the temperature sense may also be affected. The reflexes in the parts affected are diminished or lost. The electrical reactions are diminished and altered qualitatively.

The disease runs in families, and begins usually at a very early age, so that clubbing of the foot may be well marked at the age of five years.

The **diagnosis** must rest upon the presence of double club foot, which is not congenital, upon the sensory disturbances, and upon the slow onset and progressive character of the atrophy. As has been said, the primary lesion is apparently in the nerve trunks, although it is probable that this view might require modification in the future.

Primary muscular dystrophies occur under several clinical forms, which all agree in that, with rare exceptions, they commence in childhood or youth, and are in many instances family diseases—that is, they are hereditary, or affect several members of the same family. They differ in their point of origin, and the distinction between the clinical types depend in part upon this. Thus the atrophy may begin (*a*) in the face and extend after a time to the shoulder girdle and upper arm—the *fascio-scapulo-humeral* type (Déjerine-Landouzy); or (*b*) it may begin in the shoulder girdle and extend to the upper arm and, finally, to the lower limb—the "*juvenile muscular atrophy*" of Erb; or (*c*) it may first attack the muscles of the lower limb or pelvis—the "hereditary muscular atrophy" of Leyden and Moebius, and pseudo-hypertrophic muscular paralysis. They differ also in the effect of the disease on the bulk of the muscles. In some forms, pseudo-hypertrophic paralysis as a rule, and juvenile muscular atrophy as an exception, the bulk is obviously increased owing to overgrowth of the connective and adipose tissue. The occurrence of true hypertrophy of the muscular fibre is denied by some authorities, but it is probable that in some (juvenile form of Erb), and possible that in all, it is the first change which occurs. Next there is proliferation of the connective tissue, with or without deposit of fat, and finally there is complete disappearance of the muscular tissue, though the bulk of the muscle may be maintained or, as in pseudo-hypertrophic paralysis, greatly increased. Eventually, however, even in this type, the fat is absorbed, and the atrophy of the muscles becomes evident to the eye. Long before this the atrophy of muscular fibre is shown by loss of power and by the disappear-

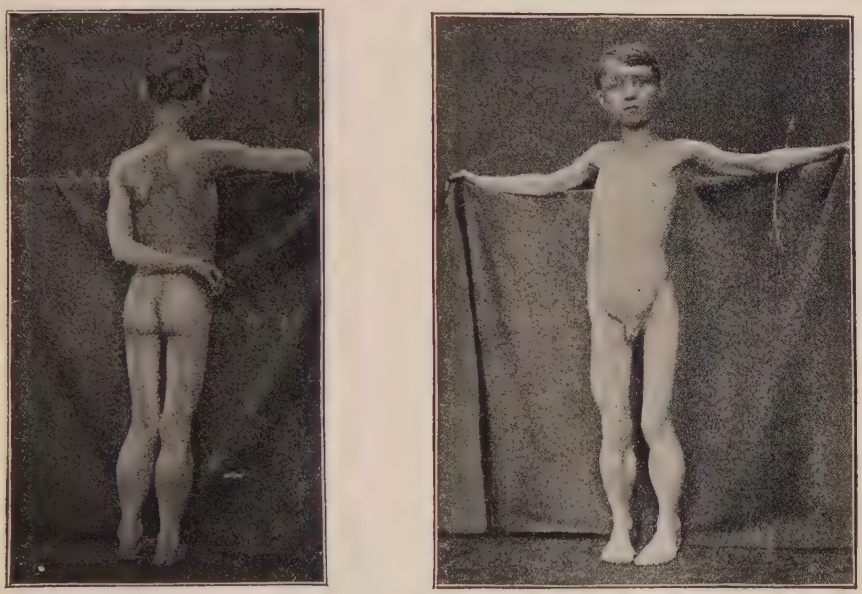
ance of mechanical and electrical irritability and of tendon reflexes. The reaction of degeneration is not present, and there are no fibrillary twitchings.

The **pathology** is obscure. There is no evidence of any change in the cord, and the suggestion that the primary defect is a trophoneurosis appears to be negatived by the fact that in the same muscular bundles some of the fibres may be atrophied, others perhaps hypertrophied, and others unaltered. If the change was a trophoneurosis it would be reasonable to expect all fibres innervated from the same cell or group of cells to suffer alike. Further, the distribution of the paralysis is not the same as in disorders known to be due to spinal lesions. The fact that the atrophy affects commonly several members of the same family in the same or in succeeding generations, and the further fact that it begins in early life, lend support to the view that the primary defect is in the muscle fibres. While the nerve-cell and nerve are derived from the ectoderm, the muscle fibre is derived from the mesoderm and may develop without structural abnormality in the absence of any nervous connections. Though the life of a muscular fibre appears to depend on the integrity of its connection with a healthy multipolar cell, this, obviously, does not preclude the possibility of its presenting some inherent vice of constitution.

Pseudo-hypertrophic muscular paralysis is the best known and probably the most frequent type of myopathic atrophy. It affects males four times more frequently than females, but when hereditary transmission can be traced, it is through the mother. The disease may manifest itself at any time after infancy, in one-third of the cases when the child first attempts to walk, in one-third between the fourth and sixth year, and altogether in three-fourths before the tenth. The parents notice that the child, who has, perhaps, learnt to walk late, walks clumsily, often falls, and has great difficulty in getting upstairs. Enlargement of muscles is seldom noticed in children under five. The affected muscles are not only large but extremely hard, and do not become much softer when relaxed. The enlargement is commonly first seen and is most conspicuous in the calves. The extensors of the knee (rectus and vasti), the glutei, and the lumbar are often enlarged. Of the muscles of the upper limb the infra-spinatus is most often enlarged, the deltoid sometimes, and the muscles of the arm in diminishing degree from above down. The latissimus dorsi and the lower part of the pectoralis major do not enlarge, but, owing either to atrophy or failure in development, are often absent when the case is first seen. The absence of the posterior fold gives a peculiar appearance to the axilla. Most of these points are illustrated by the photographs reproduced in Figs. 71 and 72. As a rule, the muscles of the neck and face escape. The degree

of muscular palsy is not directly related to the degree of enlargement. In the early stage of the developed disease the child rises from the ground in a characteristic way. He first gets on his hands and knees, then he spreads the hands and knees as far apart as possible, throwing the weight on the hands; then, getting the toes on the ground and swinging the body back, he gets the knees extended, and "walking" the hands along the floor throws part of the weight of the body on the legs; lastly placing one hand on one knee, he pushes with the other off the ground, throwing the weight

FIG. 71. FIG. 72.



Pseudohypertrophic paralysis, showing "hypertrophy" of some, and atrophy of other muscles. (From a photograph by Dr. PERCY C. PHILLIPS.)

of the body back and so extending the hip. At the same time he supplements the advantage thus gained by pressing with the hands, which are shifted alternately up the thighs.

The order and degree in which the muscles of the lower limbs are affected appears commonly to be—flexors of hip (psoas and iliacus), extensors of knee (rectus and vasti), extensors of hip (gluteus maximus mainly).

The difficulty in getting up stairs is due to the weakness of the extensors of the hip and knee. In walking, the pelvis oscillates widely, being tilted with each pace so as to bring the centre of

gravity over the foot which is on the ground. This is done owing to the weakness of the hip muscles, and in particular because the gluteus medius is too weak to counteract the tendency of the pelvis to tilt towards the side on which the foot is off the ground. Another effect of the weakness of the extensors of the hip is lordosis on standing, owing to the pelvis being tilted forward; to bring the centre of gravity over the feet, the upper part of the trunk is carried backward, so that a line dropped from the scapular angle falls well behind the sacrum. When the child sits down the lordosis disappears, and is replaced by curvature in the opposite direction, owing to the weakness of the spinal extensors. The peculiar manœuvers practised in rising from the ground are designed mainly to replace the diminished power of the extensors of the knee. When these muscles are entirely destroyed the patient cannot rise or stand. Increasing weakness of the muscles of the back renders him unable to sit up, and when placed in the sitting posture the trunk bends forward, producing extreme posterior, often combined with some lateral curvature. The shortening and atrophy of muscles lead to various deformities of the limbs, commonest and earliest among which is talipes equinus, due to the shrinking which succeeds the overgrowth of the calves.

The **prognosis** is bad, for the child usually succumbs, at about or before the age of puberty, to bronchitis or some other intercurrent malady. The duration of life is likely to be longer the older the patient is when the disease is first observed. In a few cases the disease appears to have been arrested—at any rate for some time; and the prognosis is on the whole better in girls than in boys.

The **diagnosis** of the disease is generally not difficult—the age of the patient, the progressive loss of power, the peculiar gait the lordosis on standing, and the peculiar manner of rising from the floor, will suggest the disease, and the detection of enlarged and hardened muscles (especially the calves and infra-spinati) will confirm the diagnosis.[1] The distinction from other types of myopathic atrophy depends on the distribution of the palsy and the enlargement of the muscles, but cannot always be made. The absence or great diminution of the knee-jerks, the peculiar way of rising from the ground, the gait, and the passive nature of the contracture ought to prevent the disease being confounded with spastic paraplegia.

Treatment can do nothing to arrest the progress of the disease. Arsenic and phosphorus have been thought to have some beneficial effect, but their influence is doubtful, since the disease, though on the whole progressive, may spontaneously present intervals of arrest or much retarded progress. Electricity may be of some service in

[1] Enlargement of the infra-spinatus, with disappearance of the latissimus and the lower part of the pectoralis, Gowers regards as almost pathognomonic.

stimulating growth, and massage in improving nutrition, but the voluntary stimulus is the most effectual. Gymnastic exercises are therefore to be recommended. The contraction of the calf muscles, and the consequent equinus which may prevent the patient from walking at an early stage of the malady, may be remedied by tenotomy.

CHAPTER XLVI.

DISEASES OF THE SKIN.

Urticaria—Urticaria Papulosa—Raynaud's Disease—Prurigo—Urticaria Pigmentosa—Erythema Simplex—Erythema Intertrigo—Erythema Scarlatiniforme—Erythema Multiforme—Purpura—Peliosis Rheumatica—Chilblain—Pemphigus—Herpes—Pruritus—Itching; Pediculosis; Scabies.

Angeio-neurosis.—The cutaneous structures are liable to vaso-motor disorders in which the mucous membranes and, perhaps, certain of the viscera may also share.

Urticaria is the most common and typical example. Under the influence of various causes, of which the most important and frequent are digestive disturbances, a localized œdema of the skin develops rapidly owing, probably, to paralytic dilatation of the arterioles. The resulting swelling, or wheal, has a white centre with a red border. As the œdema subsides the centre becomes red, while the white color extends to the edge. The size and number of the wheals vary very much. They may be large, and then generally few in number, or numerous and small. Their appearance is attended by a great deal of itching and discomfort, and the child is often a little feverish. The wheals when small are often very evanescent. When large they usually last a few hours, and then fade, leaving a slight redness of the skin, which disappears in a few hours. When at their height the wheals are firm, but if placed on or near parts with much loose connective tissue, such as the eyelids or scrotum, there may be extensive œdema, producing much deformity of the part. In rare cases no distinct wheal forms, but a more or less extensive surface becomes œdematous. The face, lips, cheek, hands, or legs may be attacked. The œdema is tense, and there may be some itching before it is fully developed. Sometimes the attacks recur periodically and even at the same hour on many succeeding days. The tendency to this condition occasionally runs in families. In many cases the outbreak is attended by colic and other symptoms of gastro-intestinal disturbance. As a rule the child ceases to be troubled by the attacks after some years, but the liability may continue for an indefinite period, and death has been produced by sudden œdema of the larynx. In children the most important form of urticaria is that to which the term **urticaria papulosa** has been applied by Colcott Fox. The con-

dition, which was formerly called lichen urticatus, causes very great distress both to mother and child, since the irritation it produces prevents sleep and keeps the child continually restless—"always on the fidget." The individual wheals are small and at first evanescent. After a time there are, mixed with the ordinary wheals, others with a hard central papule, which does not disappear as the wheal subsides, but remains as a red point when the color is discharged from the rest of the wheal by pressure. The papules itch intensely, and by scratching become infected. They then become crowned by small pustules, which are succeeded by scabs. The papules appear on all parts of the body, especially upon the trunk, forearms, and calves. In severe cases the child or infant may be covered almost from head to foot with papules, pustules, and scabs.

Among *causes* of urticaria, the first place may be given to local irritants, since the disease owes its name to the nettle, which produces typical urticaria on a small scale. Many insects which attack man also produce local urticaria. The most important causes, however, are poisons derived from the gastro-intestinal canal. These may be ingested. Mussels and other kinds of shell-fish, for instance, invariably produce an attack in some persons; in other cases, the poisonous quality of the food is due to some change of the nature of decomposition, which has taken place in it. In other cases, and these are in practice the most important, the toxic substances are produced during digestion, owing to some defect in that process. Urticaria papulosa is, in many cases, associated with dilatation of the stomach; in others, with chronic and intestinal catarrh; but, in others, the infants are well nourished, and present no symptoms beyond, at most, a little flatulent dyspepsia. In fact, in all forms of urticaria dependent on digestive disorder, idiosyncrasy plays a very large part. Urticaria papulosa may occur at any time of the year, but is usually worse in warm weather. In many, perhaps the majority of cases, attacks recur again and again for years, and, according to Malcolm Morris, may be the first stage of true prurigo.

In the **treatment** of urticaria papulosa, the most serious form of urticaria in the young, the first indication is to allay the itching and prevent scratching. A warm or rather hot bran, starch, or alkaline bath should be given, or a plain bath with the use of superfatted menthol soap, and the body should be quickly dried by dabbing with a soft towel.

[*Bran Bath.*—Take 2 oz. bran for each gallon of water, enclose it loosely in a muslin bag, and allow it to soak for 10 minutes; stir the water with the bag, and remove.

Starch Bath.—Potato starch, $\frac{1}{2}$ oz. to each gallon of water.

Linseed Bath.—Linseed meal, $\frac{1}{2}$ oz. to each gallon of water.

Alkaline Bath.—Sodium bicarbonate, $\frac{1}{4}$ oz. to each gallon of water.

About 1 drachm of borax for each gallon may be added with advantage in many cases. Appendix.]

The garment put on next the body should be of fine cotton, and should be well powdered with starch powder. If the itching returns quickly, as it commonly does on exposed parts of the body, these should be dabbed with an antiseptic and sedative lotion, or with calamine lotion, or an evaporating lotion.

[Lotio Calaminæ—

Calamin. Præparat.,	gr. xl
Zinci Ox.,	gr. xx
Glycerin.,	℥
Aq.,	ad ℥j. Appendix.]

If the papules are suppurating on the surface, or covered with blood or crusts, the parts thus affected should be treated with an antiseptic ointment, for which purpose nothing is better than a weak sulphur ointment. Equal parts of sulphur and zinc ointments and vaseline make a good application, or the zinc ointment may be replaced by carbolic acid ointment. A simple boric acid ointment with a basis of equal parts of lanoline and olive oil is also a useful application, to which cocaine (gr. x to vaseline ℥j), or carbolic acid (gr. xx to ℥j), or both, may be added. The most important point is the treatment of the attendant gastro-intestinal disturbance. A dose of castor oil should be given at once and followed by saline aperients for several mornings, or by a castor-oil mixture if the stools contain much mucus. The best results are obtained by long-continued use of intestinal antiseptics, especially calomel or salol. To allay the itching produced by mosquitos, bugs, and other insects, ointments containing cocaine, carbolic acid, or ichthyol will be found useful. Urticaria due to ingesta must be treated, if the case is seen early enough, by giving an emetic, and in any case by a brisk purgative. Occasionally urticaria produced by shell-fish is attended by serious general symptoms of nervous depression, and in such cases the stomach should be washed out without delay and a purgative given.

Raynaud's disease is characterized by capricious attacks of defective circulation in parts—fingers, toes, ears, and nose—most remote from the centre of the circulation, and most exposed to the influence of cold. It is due to vaso-motor disturbance, which produces first contraction and then paralytic dilatation of the small arteries and arterioles. The disease may commence as early as the end of the second year, and the attacks usually recur many times in each winter, the patient being free during summer weather.

The **symptoms** vary in intensity. After exposure to cold, after emotional disturbance, or without obvious cause, the fingers or toes, the parts most often affected, become "dead," cold and pale. This

stage of local syncope is followed by reaction, during which the fingers are hot, red, and tingling; or by local asphyxia, in which the affected parts become intensely congested, œdematous, cold, and deep red or purple in color. In the former case there is a true reaction with increased flow of blood through the parts; in the latter, almost complete arrest of the capillary circulation, with venous congestion.

Local asphyxia may be so intense and persistent that *gangrene* ensues. It is usually more or less symmetrical, and affects, especially, the tips of the fingers and toes, the edge of the ears, more rarely patches of skin on the limbs or trunk. These severe attacks are accompanied by acute pain, but, as a rule, though the local asphyxia may involve nearly the whole of the hands and feet, the part which actually becomes gangrenous is small. In rare cases gangrene may rapidly involve hands and forearms, feet and legs. Ankylosis of the phalangeal joints has occurred, and peripheral neuritis has been observed. In some cases internal organs are affected, either during the attacks of local asphyxia or alternately with them. Thus transient hemiplegia has been recorded, and paroxysmal attacks of hæmoglobinuria (*q. v.*). The general symptoms which accompany the attacks are not characteristic. There is no fever, but the patient feels depressed, has no appetite, and occasionally suffers from delusions. Children who have Raynaud's disease are generally ill-nourished and rather dull, especially during cold weather.

The **treatment** must consist mainly in warding off the attacks by avoiding exposure to cold, and by keeping the patient warmly dressed. During the attacks the affected parts should be wrapped in cotton-wool; in severe attacks the child should be kept in bed, and in the most severe it may be necessary to give morphia to relieve the pain. Moebius has suggested that a course of treatment by thyroid gland might be of use; massage benefits some patients. Barlow advises the use of a galvanic bath for the limbs, one electrode being over the spine and the other in the water, which should contain some common salt.

Prurigo is a chronic papular eruption attended by the most intense itching. It begins usually during the first year of life, when it is practically indistinguishable from the much more common urticaria papulosa. The papules, which are very persistent, are eventually attended by a fibrous degeneration of the cutis due to long-lasting chronic inflammation. The papules occur in greatest number on the extensor surfaces of the limbs, especially of the legs, and considerable glandular enlargement may ensue. Owing to the itching the patients scratch violently, and various impetiginous lesions are usually to be found mixed up with the papules and obscuring the nature of the affection. The disease is very obstinate, but is liable

to remissions and can be relieved by treatment, which should be of the same kind as that recommended for urticaria papulosa. The patients suffer a good deal in general health and nutrition, and need a nutritious diet, cod-liver oil, and preparations of iron.

Urticaria pigmentosa is a rare affection, allied to urticaria papulosa, but differing in the character and distribution of the lesions and in its course. During the first few weeks, or, at latest, within three months of birth, urticarial patches of varying size, but reaching sometimes the diameter of half an inch, appear. They are at first conical and red, but after some days become flattened and of a brown color. Others appear in succession and pass through the same phases. The distribution is usually symmetrical, the parts chiefly affected being the front and axillary areas of the thorax, the limbs, and the belly. Owing to the intense itching which attends the patches, various inflammatory lesions of the impetiginous type are apt to be produced by scratching. During the second year the spread of the disease becomes arrested, after the age of five or six years improvement begins, and recovery is generally complete in two or three years more. No treatment appears to be able to check the course of the disease, and all that can be done is to attend to the general health and nutrition, to relieve itching, and to treat complicating impetigo by the ordinary methods.

Erythema signifies, properly, redness of the skin due to hyperæmia of its more superficial parts, but the term has been extended to embrace a number of other conditions in which hyperæmia is an early or prominent symptom.

Erythema simplex (patches of redness, seen usually on the face, or on the folds of the neck, axilla, groins, or buttocks) is extremely common in infants and young children. The part is at first bright scarlet and hot, and there are sensations of burning and itching. The color fades to pink, and some slight desquamation and very superficial yellowish staining of the skin may attend subsidence of the erythema. In some cases it appears to be due to reflex irritation from difficult dentition, intestinal worms, or gastro-intestinal irritation. In such cases it is usually fugitive, lasting perhaps only a few hours, but very apt to reappear either in the same or some other situation; the term **erythema fugax** is applied to such cases. The erythema produced by exposure to the sun, or to the direct influence of cold winds, belongs to this class.

Erythema involving folds of skin in contact with each other, **erythema intertrigo**, is a more obstinate affection, and, owing doubtless to the retention between the opposed surfaces of irritating secretions, is very apt to be complicated by true superficial inflammation, attended by weeping, which is absent in simple intertrigo. It sometimes extends over the abdomen, involving all the parts

covered by the napkin, and may then raise a suspicion of congenital syphilis. The fact that in syphilis the erythema is more widespread, extending down the posterior aspect of the legs and to the soles of the feet, and that the skin is usually a little thickened and has a brownish or purplish tint, taken together with a consideration of all the circumstances of the case, will usually prevent error. It is, however, necessary to be on one's guard, and in cases of obstinate erythema of the buttocks to make careful search for other evidence of syphilis, which will seldom be altogether wanting.

In the **treatment** of erythema simplex the main indication is to discover and remove the cause. The part should be dusted, or dabbed with calamine lotion. If the erythema be limited to the area covered by the napkin, it is probable that the napkins are not sufficiently washed, or that some irritating material is used by the laundress and not rinsed out, or that the napkin is not changed often enough. Intertrigo should always receive prompt attention. The parts should be kept scrupulously clean, using a weak boric acid solution, and avoiding soap as much as possible, and well powdered, and should be kept apart by pledgets of absorbent cotton. [Dusting powder—zinc oxide, boric acid, starch powder—equal parts.] A muslin powder bag is a convenient application, but is not well suited for the buttocks and groins in infants, as it is very apt to become soiled.

Erythema scarlatiniforme is the term applied to the erythematous eruption which accompanies certain simple febrile affections, especially that form of pharyngitis or tonsillitis which is produced by exposure to the emanations from foul drains. In other cases the rash occurs in the course of pneumonia, diphtheria, or septicæmia. It has been observed also as a complication of rheumatism, malaria, and syphilis, and has then been attributed by some to the toxic action of mercury, sodium salicylate, or other drug which has been administered. In a minority of cases no cause can be discovered, and in some individuals the disease shows a tendency to recur every spring, or at irregular intervals. Erythema of this character occasionally follows the administration of an enema. Not infrequently the erythema is more patchy, and a little resembles the eruption of measles (rubeoloid erythema). This type of erythema is due probably to the absorption of toxic bodies from the intestines, and erythema scarlatiniforme is probably, in all cases, a manifestation of a toxæmic condition. *Treatment* therefore must be directed to the condition of the throat should it be inflamed, or of the stomach and intestines. It should be commenced by the administration of a laxative dose of calomel, unless special conditions exist contraindicating the use of the drug, when its place may be taken by castor oil. The sanitary condition of the house should be inquired into, especially in those cases in which relapses occur. Even when no very marked gastro-intestinal symp-

toms exist, regulation of the diet and the use of stomachics and intestinal antiseptics should be resorted to.

Erythema multiforme is an inflammatory disease of the skin only occasionally seen in children, and, in them at least, nearly always a manifestation of the rheumatic state. Its onset is attended by marked constitutional symptoms, fever, pains in the joints, sore throat, and diarrhœa. The rash, which is attended by some pain and burning, but not by much itching, appears as a rule first on the dorsal aspect of the hands and feet, and is distributed more or less symmetrically. Subsequently it extends to the forearm, thighs, and trunk, covering sometimes very large areas. Beginning as an erythema, nearly every form of lesion of the skin—papules, vesicles, bullæ, petechiæ may subsequently develop. On the whole the affection of the skin in any area tends to heal first at the points at which it appears first, so that concentric circles and intersecting rings of eruption after a time encircle, or surround in an irregular manner, patches of healthy skin. The duration is very uncertain ; an attack lasts usually several weeks, but as relapses are common the course of any case may be very much more prolonged. In rare cases the general symptoms are very severe, and the occurrence of pericarditis or endocarditis practically carries the case into a different category. Endocarditis is often of the malignant (infective) type, and the prognosis correspondingly serious. **Treatment** has not much effect on the course of the disease even in its milder forms. Sodium salicylate should be given at the onset for a day or two ; later quinine should be given, and small doses of opium may be required. After the disease has ceased to extend rapidly, arsenic is useful. Locally, calamine lotion or compresses wrung out of a weak carbolic lotion may be used to relieve the discomfort. The general management of convalescence should be the same as after rheumatic fever.

Purpura, extravasation of blood into the superficial parts of the skin, as indicated by purple spots, streaks (vibices), and patches—small (petechiæ) or large (ecchymoses)—may be the result of any condition causing intense hyperæmia of the skin, and is thus occasionally a consequence of erythema. In practice among the poorer classes the commonest cause of petechiæ is flea-bite. Marasmic infants and young children may often be seen covered with petechiæ, most abundant about the shoulders and chest, due to this cause. With a lens the central puncture may usually be made out.

Peliosis rheumatica is an acute disorder characterized by purpura and joint pains. It occurs usually in patients who have already suffered from various rheumatic manifestations. It is not common in childhood. The onset is marked by general malaise, which is accompanied by a rise of temperature. The joints then become painful, red, and swollen, and in a day or two the rash appears about the

wrists, knees, and ankles. It consists of red patches, which may be slightly raised, and do not fade on pressure; their color deepens quickly and finally becomes dark purple or black. As the rash comes out the joint pains abate, and the patient is convalescent in a few days unless the heart be involved, as is sometimes the case, or unless the rash occurs as a complication of distinct rheumatic fever. Even in the slightest cases, however, a relapse is very likely to occur after a week or two, and in some cases many such relapses succeed each other, so that the whole illness lasts several months. The patient should be kept in bed during the onset of the disease, and at each relapse. Sodium salicylate produces the same kind of alleviation as in other rheumatic affections. During convalescence the patient should have a nourishing diet and as much fresh fruit as can be taken without inconvenience. Iron preparations are also to be recommended, as a rule.

Chilblain, to which the term *erythema pernio* has been applied, not very happily, is in reality an acute inflammation of the whole substance of the skin. It is a very common affection in children, especially those who are growing fast, and are "big for their age." It runs in families, and is perhaps most common in neurotic children and in those of "scrofulous" type. It affects chiefly the hands, feet, and ears, parts which are exposed and are farthest removed from the centre of the circulation. When the circulation is poor, as, for instance, in a paralyzed limb, the liability to chilblain is greater, and large tracts of skin may be involved. The disease comes on usually during weather which is both cold and damp. A child who has been almost free during a hard frost will begin to suffer severely during the succeeding thaw. Once established, the liability to relapse is very marked; recurrence is also the rule, so that the patient suffers during each succeeding winter, from early childhood until adult age. Chilblain is an extremely distressing disorder, owing to the intense itching and aching which attends the acute stage. If neglected at this stage a blain, or large shallow bleb, forms. This is easily ruptured by scratching or by friction of the clothes, and we then have a condition in which a shallow but very vascular ulcer rests upon and is surrounded by skin in a condition of acute inflammation. The part in this stage is extremely tender, and the child dreads even a light touch, so that it avoids games and desires to keep still. Severe chilblain on the feet, in fact, render walking practically impossible. Occasionally beneath the blain, or even before it has formed, necrosis occurs, and a deep ulcer with sharply cut edges results. This has no special characters and is not exquisitely tender, as is the form described above.

The **prophylactic treatment** of chilblains is a matter of considerable importance, since when once developed they are extremely obstinate,

and if severe, prevent the child taking exercise, joining in school games, and by the constant irritation and interference with sleep produce a condition of great nervous irritability and a general deterioration of the health. The most important precaution is to see that the child does not wear damp clothes. During a thaw and at other times when the air is near saturation point, all clothes, but especially woollen garments, and boots and shoes, readily take up a large quantity of water. The use of such garments, but especially damp gloves and boots, keeps the parts constantly cold, and undoubtedly favors the production of chilblains. If the child is old enough it should wear woollen vests and drawers, knitted woollen armlets and stockings, thick-soled shoes (not boots) and cloth gaiters. Every garment should be well aired; gloves, stockings and boots should be taken off as soon as the child comes in and replaced by aired garments. If the cold morning bath is given—and it should be replaced by a hot bath if reaction is not good, or if the child shows dread of cold water—it should be followed by vigorous rubbing with a flocculent towel in a warm room. The child should be taken out of doors as much as possible, but should be made to take exercise and not allowed to dawdle about. The diet should be plain and ample, containing a full proportion of proteids. As a rule, alcoholic beverages should not be allowed.

The **treatment** when once the chilblain has formed must depend upon its stage. In the earliest erythematous stage the greatest relief is obtained by plunging the part into hot water, and gradually raising the temperature by adding hotter water. After such a bath of ten or fifteen minutes' duration, the congestion is very much diminished. The part should then be thoroughly dried with a soft towel, smeared with belladonna and glycerine, and packed with absorbent cotton-wool, retained in place by a bandage applied firmly. This should be done the last thing at night and twice or thrice during the day. If the chilblains are small they may be painted with tincture of iodine, which has a gently astringent action. Itching is relieved by compound tincture of benzoin and by camphorated spirits, but best by a cocaine ointment. The application of collodion with the idea of exerting pressure on the chilblain is usually worse than useless. The collodion cracks with the movement of the part, and in each crack a shallow linear ulcer forms. A somewhat similar objection applies to iodine if used in too strong a solution. When a bleb has formed it should be dressed with a simple antiseptic ointment of boric acid to which a little cocaine is added, or of carbolic acid, and should be protected from injury. The painful vascular ulcer should be treated with an antiseptic ointment and continuous hot fomentations or poultices. It is absolutely necessary to give the part rest, and if the feet be the part affected, the child must be kept in bed or

on the sofa for a day or two. When once the surrounding dermatitis has subsided the shallow ulcer will heal quickly. Ulcer succeeding sphacelus must be treated on ordinary surgical principles. Internal remedies have little or no effect, except, perhaps, preparations of iron, especially the perchloride when it can be borne. If not, the syrup of the phosphate may be given. Cod-liver oil should not be given unless other indications exist for its use.

Pemphigus is a term often used very loosely ; it should be confined to those cases in which, with or without slight antecedent erythema of the area to be affected, a bulla appears, rapidly attains its full size, and in the course of a day or two dries up, leaving a dark yellowish scab, under which the skin, covered by delicate epithelium, has a bluish color. The color changes to brown, and no scarring results. The mucous membranes are attacked occasionally. The etiology of pemphigus is obscure. In some cases an hereditary tendency exists. In others, the disease apparently has a septic origin, and the lesions are probably due to toxæmia (see " Pemphigus Neonatorum "). The bullæ appear at first in crops on various parts of the limbs, trunk, and face (lower part), and there is fever and some general disturbance of the health. Occasionally hæmorrhage takes place into the bullæ. More often, owing to scratching and want of attention, suppuration occurs, usually after rupture of the bleb, and ulceration ensues. In feeble children the presence of numerous bullæ, especially if suppuration ensue in connection with them, produces great exhaustion. Independently of discoverable infection, the intensity of the local lesion may cause gangrene and sloughing. Except in cases of this type, recovery after a few weeks is the rule, though there is considerable liability to relapse and recurrence.

Though usually chronic or subacute, pemphigus is occasionally, especially in young children, very acute (malignant). The number of bullæ is very large, and appear in rapid succession. Fever does not disappear soon after the onset, as is the rule, but is continuous, and the patient's strength is rapidly exhausted, and death occurs in a week or two.

The **diagnosis** is not always easy unless the case can be watched or a thoroughly trustworthy history obtained. True pemphigus is, in my experience, a rare affection in childhood. The great majority of the cases to which the term is commonly applied are examples of pyococcal infection in which the inflammation spreads with great rapidity immediately beneath the epidermis and thus produces large blebs, which rupture or dry up before obvious suppuration occurs.

In the **treatment** of true pemphigus the main indication is the administration of arsenic. A small dose should be given at first, and increased more or less rapidly according to circumstances. Quinine is also a valuable remedy, and should be given at the same time.

Failing arsenic, phosphorus or belladonna should be tried. Locally, the condition should be treated by antiseptic ointments, and the blebs should be guarded from rupture. If very tense, they must be punctured with a sterilized needle, and dressed with a mild antiseptic ointment. The general strength should be maintained by placing the patient under the best obtainable hygienic conditions, taking him into the open air when possible, and administering a copious simple diet.

Herpes is the term applied to a vesicular eruption which occurs under two very different conditions, though in both the arrangement of the vesicles appears to be governed by the distribution of the nerves of the part. The characteristic lesion is a cluster of small vesicles, which form upon a limited area which has for some short time previously been hot, swollen, tense, and painful. The contents of the vesicles become opaque, often purulent, and eventually a yellowish scab forms, which finally is detached without in most instances leaving any scar.

Symptomatic herpes is exceedingly common in infancy and childhood. It occurs usually as a complication of coryza, or pneumonia, and runs through its several stages rapidly, though not infrequently it is succeeded by local impetigo. It affects usually the lip (herpes labialis), most often near the middle line. Very rarely in children does it occur on the genitals. The only treatment required for the local condition is the application of a mild antiseptic ointment.

Herpes zoster is exceedingly rare in young children. As the age of puberty is approached it becomes far from uncommon. Its causes, symptoms, and course in children do not differ from those of the same condition in the adult.

Pruritus—that is to say, reflex itching, without discoverable local cause to account for it—is extremely rare in childhood, except at the nasal and anal orifices, and in the external auditory meatus. Pruritus ani or vulvæ is usually due to intestinal parasites, to hæmorrhoids or polypus of the rectum, or to retained masses of hardened fæces, and cases when the rectum and large intestine have been treated effectually. Itching of the nose, which causes the child to be constantly picking and scratching at it, appears to be associated with irritation somewhat higher up the intestine, and is especially common in children infested by ascaris lumbricoides. It is, however, sometimes due to chronic rhinitis and naso-pharyngitis. Pruritus of the ear is usually a symptom of middle-ear disease (see "Otitis"), sometimes of naso-pharyngitis.

Itching is a symptom common to many forms of skin disease, and owing to the irresistible desire to scratch, it is the indirect cause of complications by inflammation due to pus-forming micro-organisms. Itching of the head should raise the suspicion of pediculosis, of the body generally of lice, fleas, or bugs; of the hands and feet, of scabies.

The first step in treatment is to remove the cause. When **pediculi capitis** are present, they will be found in largest numbers in the occipital region. They must not be assumed to be absent because the patient has clean clothes and is well cared for. The hair should be cut as short as the parents will allow; if impetigo be present the cutting of the hair should be insisted on. The hair should be washed with hot water and a little soft soap, or with soap spirit, and combed out,—after it has dried it should be wetted with acetic acid lotion, which has a solvent action on the glutinous matter by which the nits are fixed to the hairs. Subsequently a mild sulphur ointment should be used as a pomade. When the number of nits is not very large, this ointment, combined with daily combing, will be sufficient. A lotion which has long been popular at the Shadwell Children's Hospital contains mercury perchloride, acetic acid, turpentine, and carbolic acid.

[Lotio Hydrargyri c. Acido Carbolico (Shadwell)—

Liq. Hydrarg. Perchlor.,	℥xx
Acidi Acetici Dil.,	℥xl
Ol. Terebinthinæ,	ʒij
Sol. ac. Carbol. (1 in 40),	ad ʒj. Appendix.]

It is an example of poly-pharmacy, but it is very useful with dirty people who will not take much trouble. Body lice are, for some reason (possibly because their clothes are more often changed and washed), rare in infants and not very common in children, even among the poorest classes.

Scabies is probably neither more nor less common in children than in adults. In infants it is sometimes met with on the head and face, but its seat of predilection is on the webs between the fingers and toes. With these exceptions it presents no peculiarities in early life. An eruption, most marked on the fingers, the back of the hands, the toes and the dorsum of the foot, should raise a suspicion of scabies. It should be remembered that the itching produced may extend far beyond the actual seat of the primary lesion. Suppuration quickly ensues in and about the burrows in children who are not very clean, and the purulent infection is rapidly carried by the finger nails to distant parts, the seat of reflex itching. The front of the trunk, the back, and indeed, every part of the person, may thus become covered with suppurative lesions in various stages.

In the **treatment** of scabies in children the ordinary method should not be too vigorously applied, as it is easy to stir up an excessive amount of general irritation of the skin. As a rule, treatment may be commenced by giving a warm bath with soft soap, which removes the superficial dermatitis and lays bare the burrows. Sulphur ointment (precipitated sulphur, gr. xx–xxx to ʒj of lard or

equal parts of lard and vaseline, with a little essence of lemon to cover the odor) should then be rubbed in, at first four times a day, afterwards once a day. If the infection be confined to the hands or feet, and accompanied by much crusting from suppurating lesions, the parts should be first soaked in carbolic oil for a day or two, after which the washing and sulphur treatment may be commenced. In the same way, when extensive impetiginous lesions are present in many parts of the body, treatment should be commenced by a mild sulphur and zinc ointment, and when the impetigo and other suppurative lesions have begun to subside, the special treatment may be commenced. The clothes should be stoved or boiled.

For the relief of *itching*, of which the cause cannot at once be removed or discovered, various local sedatives and anæsthetics may be used. A warm bran, starch, or alkaline bath is often very soothing, as is also a hot bath with a non-irritating soap. Lint steeped in vinegar and water (equal parts), or in a simple spirit evaporating lotion, and applied to the part where the itching is most intense, gives temporary relief. A saturated solution of menthol in spirit painted on produces a grateful sense of coolness. Carbolic acid, or menthol, or cocaine in a lanoline ointment base, has a more lasting effect, and the first named will sometimes have a curative effect by removing the cause. Occasionally salicylic acid (gr. x to ʒj) has a good effect for the same reason.

CHAPTER XLVII.

DISEASES OF THE SKIN—(*continued*).

Pyococcal Dermatitis—Impetigo—Catarrhal Dermatitis—Furuncle—Ringworm—Alopecia Areata—Seborrhœa—Lichen—Miliaria—Eczema and Psoriasis.

Dermatitis due to infection by pus-forming organisms, owing to the frequency with which it occurs, overshadows in practical importance all other forms of skin disease in childhood. Not only does it occur as a primary affection, but it is very liable to complicate almost every other form of skin disease, especially among those classes of the people who are disposed to neglect the earlier manifestations of disease, and by whom cleanliness is little regarded. The infective agent is, as a rule, one of the pyogenic staphylococci (aureus, citreus, or albus), but sometimes the pyogenic streptococcus. The lesions produced by the latter are, as a rule, more severe and more disposed to spread by contiguity.

Three main varieties may be distinguished: impetigo, catarrhal dermatitis, and furuncle. Pyogenic organisms are usually present in various parts of the skin in health, and the opportunity to develop and to produce their characteristic lesions is afforded usually by traumatism or irritation, more rarely by some deterioration of the general health, which has diminished the resistance of the skin.

Impetigo is a pustular eruption which may attack any part of the surface, but is especially frequent on the exposed parts. The organisms present are usually staphylococci. The suppuration may be preceded by a brief vesicular stage. It may be determined by almost any source of irritation. Thus on the scalp it frequently complicates pediculosis; on the face it is often derived from the vesicles of herpes labialis which have become infected; on the hands and feet it is prone to follow any accidental scratch or abrasion which has not been kept clean; or it is secondary to scabies, or contracted by scratching some other part already infected. Self-inoculation is, in fact, one of its most striking characters. The infection may be carried not only by the finger nails but also by the clothing. Thus it is not uncommon to see a patch of impetiginous dermatitis well developed on the outer side of the malleolus on one side, and in an earlier stage on the inner malleolus on the other side, the infection having clearly been carried by the interchange of stockings. The

list of such occurrences might be multiplied almost indefinitely. The infection may, under certain favoring circumstances, among which perhaps the degree of virulence of the staphylococcus is one, be contracted from a previous case, whence the term *impetigo contagiosa*. Occasionally widespread impetigo will be found to have had its origin in a mild attack of varicella for which medical advice has not been sought. The pustules rupture and the surface becomes covered with crusts, which are at first easily detached, but subsequently become more firmly adherent. Beneath the scabs suppuration may occur.

There is a variety, due in many if not in all cases to the streptococcus, in which the inflammatory process spreads with great rapidity immediately beneath the epidermis; the fluid effused, which is at first serous or opalescent, raises the epidermis, forming sometimes more or less circular bullæ, more often irregular flattened shallow cavities, which tend to spread rapidly. In many cases the scar of the scratch by which the infection has been introduced can easily be detected. This form of creeping dermatitis occurs especially on the fingers. When the suppuration takes place under dense epidermis, as on the palmar surface of the fingers or thumb, the process causes a good deal of pain, and may be mistaken for deep-seated whitlow.

Catarrhal dermatitis, due to pus-forming organisms, may occur on any part of the body, but is specially frequent on the face and in the folds of the skin. It is often a sequel to impetigo, and near the margin a few pustules may almost invariably be found. On the face it is secondary to herpes, to stomatitis, or to suppurative rhinitis. Its most characteristic form is seen as a consequence of the last-named disorder. The secretions from the nose traversing the upper lip may produce a copious crop of impetigo, a crusting dermatitis, or a superficial dermatitis, which, if neglected, soon begins to ulcerate. A more widespread and usually less severe dermatitis is produced if in wiping the nose (often in boys of the poorer classes with the back of the hand) the purulent secretion is rubbed into the upper lip and the cheeks. The skin becomes red, then glazed, it cracks and begins to weep, leading to the formation of thin yellow crusts with, probably, some scattered impetigo at the margins of the lesion. In the folds of the skin, behind the ear, in the buttocks and groins, and, in infants, among the rolls of skin in the neck and at the ankles and knees, an inflammatory intertrigo is easily produced. The sides of the fold are red, dry, often desquamating, while at the bottom there is a collection of pustules or a shallow linear ulcer. Under unfavorable conditions of personal and domestic hygiene this ulceration may extend rapidly, causing considerable loss of substance and even placing the infant's life in danger.

In the **treatment** of impetigo the main points are to (1) prevent the extension of the disease by scratching or by wearing contaminated garments, (2) to remove crusts by soaking in oil (carbolized), and (3) to apply some parasiticide, of which sulphur is perhaps the best, though white precipitate and other mercurial preparations answer very well. The remedies are best used in ointment, and if there be much irritation of the skin, the sulphur ointment should be freely diluted and modified by the addition of zinc oxide. Salicylic acid ointment (gr. x–xv to ℥j) made with vaseline, or with lanoline and olive oil, is a valuable remedy, especially for impetigo about the face. A very useful ointment when there is much irritation consists of salicylic acid (gr. xv), bismuth carbonate and starch powder (of each ℥j), in zinc ointment (℥j). Superficial catarrhal dermatitis should be treated in the same way. In intertrigo the parts must be kept very clean by the use of antiseptics in oil or ointment, well dusted with an antiseptic powder—[Antiseptic dusting powder : zinc oxide, boric acid, starch powder, mercury subchloride, equal parts]—and the folds kept apart by pledgets of absorbent cotton or boracic lint dusted with mild boric acid powder. Ulceration should be treated with mercury perchloride solution (1 in 2,000), or with black wash, or by the careful application of calomel in powder, followed by the systematic use of a dusting powder containing about one-fourth calomel. Creeping suppuration beneath the cuticle must be treated by snipping away the detached epidermis and applying calomel or iodoform in powder, taking care that the powder reaches the spreading edge. This should be followed by the use of antiseptic ointments.

Furuncle, a localized inflammation of the substance of the skin, due to staphylococci, and originating in a follicle, or in a sweat or sebaceous gland, is probably less common in children, especially in young children, than in adults. When boils occur they do not differ in any respect from those seen in adults, and are produced by like causes. Simple furuncle must be distinguished from the condition called syphilitic furuncle (*q. v.*).

Ringworm is due to infection of the hair follicles and hairs by one of the species of trichophyton. Two chief species have been distinguished by Sabouraud. They differ in their mode of growth on artificial media, and in the part of the body which is their seat of election. (1) *Trichophyton microsporon*, which is the cause of the majority (two-thirds) of the cases of tinea capitis, has small spores and a very scanty mycelium. When it infects the hairs as well as the root-sheath it is very inveterate. Ill-defined varieties are described, but the species is almost confined to man. (2) *Trichophyton megalosporon*, which is found in most cases of ringworm of the body, has large spores and a relatively copious mycelium. Several varieties are described which infect many animals as well as man.

Trichophyton is itself capable of exciting a certain amount of inflammatory reaction, but this specific inflammation readily becomes complicated, since it affords conditions very suitable to the development of pyococci. The inflammation provoked by trichophyton in the root-sheath, when it has persisted for some time, is succeeded by a fibrous thickening which tends to occlude the orifice of the follicle, while the fungus continues to grow in its deeper part. This is one of the main causes of the inveterate character of tinea capitis when well established, for the constriction of the orifice renders it difficult so to apply remedies that they reach the bottom of the follicle, where the infective agent is most active.

Ringworm of the scalp, though a disease seldom observed after the age of childhood, is discussed so fully in works on skin diseases and on general medicine that it need not be dealt with here at length. It appears first as a small red spot having a hair follicle for its centre; the spot enlarges and forms a round red patch, slightly elevated. In the next stage the redness fades; several areas have probably run together, and we have a dry surface covered with a fine scurf and showing, either over all its area or in greater number near its margin, numerous clubbed and broken hairs, which are easily pulled out. In a stage still later the areas are less well defined, indeed almost the whole scalp may be affected. The hair is scanty, and the individual hairs are thin, dry, and brittle, but the characteristic broken stumps of the original hairs will probably all have disappeared.

Ringworm of the body begins in the same way—as a small red spot which spreads rapidly. The spreading edge is red and raised, while the centre ceases to be raised and becomes covered with a fine desquamation (tinea circinata). In some cases the inflammation does not subside so rapidly in the centre, and then red raised patches are produced. The favorite seats of ringworm of the body are the neck, face, wrists, back of the hands, and the outer surfaces of the lower extremities.

Kerion is the term applied to ringworm complicated by suppuration in the deeper parts of the skin. The suppuration begins at the bottom of the follicles, the skin is undermined and gives to the finger a boggy sensation. The pus finds its way out by the follicles which are destroyed, the hairs being extruded, so that when the suppuration subsides the ringworm also is usually cured.

In ringworm of the body and in ringworm of the scalp in the early stage *treatment* effectually applied will rapidly cut short the disease. The patch should be treated with iodine liniment, or blistering fluid, or acetic acid, which remove the superficial epidermis. Parasiticide remedies should then be applied. Of these the best are ointments of chrysarobin, salicylic acid, or a combination of the two (see Appendix), mercurials (of which oleate of mercury is the best), or sulphur.

[Ung. Chrysarobin Co. (Unna).

Chrysarobine,	5 parts.
Salicylic Acid,	2 "
Ichthyol,	5 "
Simple Ointment,	100 "]

Whatever ointment be selected, it should be rubbed in thoroughly thrice a day, using a small quantity on each occasion. In ringworm of the scalp this treatment must be preceded by epilation, and it is a great advantage to have the head shaved, so that small commencing areas may be seen and treated. If, as is too often the case, this treatment fail to arrest tinea capitis, an attempt should next be made to eradicate the disease by the use of spirit lotions containing some germicide, of which salicylic acid is probably the best.

In the majority of cases, however, the disease has already, when the case first comes under treatment, reached a stage when all local inflammatory reaction has ceased. The head should be washed with soft soap and water or spirit of soft soap (spir. saponis kalini), to remove scurf, scales, and dead hairs, and then shaved. [Spirit of soft soap (spiritus saponis kalini),—half a pound of soft soap is mixed thoroughly with 4 fl. oz. of rectified spirit strained through muslin, and scented with oil of lavender (5 drops).] The number of parasiticides recommended and used with more or less success is legion. Among these chrysarobin ointment (about 5j to 3j) is probably the best, but sulphur, white precipitate, oleate of mercury (commencing with 5 per cent.), and oleate of copper are useful. The mode of application is the main point. To smear the head once a day with ointment is useless. The hair must be short, the scalp clean, and the ointment must not only be well rubbed in but a cap fitted so that it is not rubbed off.[1]

The *prevention* of ringworm of the scalp is an important part of school hygiene. The infection is spread from one family to another mainly by the intimate contact which takes place in class, at meals, and during games. The most effectual mode of checking the spread is to put children affected with tinea capitis in a school by themselves. In Rome I saw a central school, established in an old monastery with a large garden. The children were treated and taught in this establishment until well. Failing such a special school, children with ringworm should be taught in a separate class-room, and have a separate playground.

Alopecia areata—complete loss of hair in certain areas—may be due (1) to nervous shock, a very rare event; (2) to infection by trichophyton; or (3) to infection by a special microbe, apparently a

[1] A good cap, which costs little and fits closely, may be made with a piece of gauze placed over the head, retained by a few turns of gauze bandage and consolidated by painting with Unna's gelatine, over which cotton-wool is dabbed.

small bacillus which infects the hair follicle, and grows into the hair, which becomes detached and falls out.

The diagnosis of the two varieties last named, which are not uncommon in childhood, is usually difficult; in alopecia due to the ringworm fungus broken hairs may be discovered near the margin, and the characteristic spores may be found. This variety of ringworm tends to recover spontaneously, and is therefore considered to be very amenable to treatment. In the third form there is little or no tendency to spontaneous recovery, but a cure may often be obtained by the systematic use of parasiticides, of which sulphur ointment is probably the best. Its strength should be gr. xx to ʒj at first, but it should be increased unless the amount of irritation produced is considerable.

Seborrhœa is a disease in which there is an increased production of the fatty secretions of the skin with, as a rule, some persistent hyperæmia. Seborrhœa is spoken of sometimes as though it were merely an excess of secretion, a functional disorder, but it is more than this. There can be little doubt that the skin is the seat of some infective agent, though it is not often possible to find any probable source of infection, and among the very large number of micro-organisms present in the secretions in such cases no one has been identified as the cause. As the disease is very common, and as the infective agent must be very widely diffused, it is necessary to assume some special susceptibility of the skin in those persons who contract the disorder.

The disease is of very great importance in practice, for even if we do not accept Unna's dictum that were the seborrhœa of children to be thoroughly treated eczema in adults would cease, it must be admitted that the disease can be treated effectually in children, and that if not so treated it gives rise to an exceedingly obstinate condition in the adult, which is either inveterate seborrhœa or, as others maintain, seborrhœa complicated by eczema.

Seborrhœa is a local disease of the skin, and its subjects may be, and often are, otherwise in robust health. At the same time, debilitating diseases such as the acute fevers and diarrhœa produce a certain predisposition.

Two varieties of seborrhœa may be distinguished: The dry, *seborrhœa sicca*, in which the fats with a high melting point predominate, and the oily *seborrhœa oleosa*, in which the fats with a low melting point are in excess. The first named is the form by which the scalp is most often affected, and, perhaps for this reason, that usually seen in infancy.

Seborrhœa always begins on the scalp, and when present elsewhere can be found almost always in that situation also. In a well-marked case of seborrhœa capitis in an infant the scalp is covered by a greasy,

dirty-yellow crust, which easily crumbles or scales away. The skin itself may be a little reddened, or not much, if at all, altered in color. The crust is thickest and most continuous over and about the anterior fontanelle, owing probably to the absence of friction and to the very common disinclination to cleanse this part of the head. At a later age there may be no more than a general greasy scurf of the hairy scalp, though down to the age of five or six, at least, crusts are very apt to form if the head does not receive systematic attention. The hair is scanty, lustreless, and brittle.

The oily form is that which is seen most often on the face, and that which shows the greater readiness to spread, though it may be doubted whether there is any essential pathological difference between the two forms. Indeed, if I may hazard an opinion founded upon the observation of a large number of cases, the oily form is, in children at least, due to a more active state of the infective process brought about by a greater activity of the physiological functions of the skin. On the face, trunk, and limbs the oily form produces yellowish, greasy crusts formed of the dried secretion and of epithelial scales resting on an area of hyperæmic skin, which is often surrounded by scattered papules. The hyperæmia persists after the removal of the scales, which are readily reproduced. The appearances present, therefore, a considerable resemblance to those of eczema. On the trunk and limbs the dry form produces hyperæmic patches covered by dry, whitish, or opalescent greasy scales, so that the condition may resemble psoriasis rather closely. The scales of psoriasis are drier, less greasy, more glistening, and tougher, but the diagnosis must rest mainly upon the discovery of seborrhœa of the scalp, or on a history that the scalp was the part first affected, as is always the case in seborrhœa.

In extending from the scalp to the trunk seborrhœa usually follows certain lines, which are those naturally taken under ordinary circumstances by the sweat. This applies more particularly to seborrhœa oleosa. Thus the patches may be observed to be older and more numerous at the back of the neck, and to extend in the vertebral groove to the waist, and at this level, where the expansion of the hips and the use of tight waistbands tend to arrest the sweat, a broad band of patches will often be found. The sweat descending on to the face from the scalp flows down the temples, or over the forehead to the eyebrows, by which it is directed towards the temples and ears. Patches of seborrhœa are common about the temples and before and behind the ear. Sweat descending over the forehead in the middle line escapes the eyebrows and flows down the sides of the nose into the naso-labial folds to the corners of the mouth. The alæ nasi, the naso-labial folds, the corners of the mouth, and the sides of the chin are the parts of the face most often affected.

The recognition of this mode of distribution will often be useful in diagnosis.

A seborrhœic skin is very apt to become the seat of *secondary infections* by pyococci. Thus suppuration may occur under the crusts on the scalp, and the infection may be inoculated on to the hands, face, or trunk, producing patches of impetigo, or superficial weeping dermatitis. The secondary pyococcal infection may cause adenitis of the cervical glands, and when seborrhœa, suppuration, and pediculi capitis occur together the adenitis is usually severe and often ends in glandular abscesses. In other cases seborrhœa, it is said, becomes complicated with "true eczema."

The **diagnosis** of dry seborrhœa of the scalp cannot present any difficulty, and there can seldom be any hesitation as to the oily form on the trunk since the distribution and character of the lesions are characteristic. The great similarity of the dry form on the trunk and limbs to psoriasis has already been mentioned, and the points upon which a diagnosis must be founded has been indicated.

In the **treatment** the all-important point is the thorough and persevering application to the skin itself of a suitable parasiticide. Special attention must in all cases be directed to the scalp. When it is covered by thick crusts these must first be removed. This may be done with hot water and soap, or soft soap spirit. The washing must be repeated at first daily, then as the seborrhœa improves, at longer intervals. In the neglected children of the poor the crusts are often very thick and matted together with the hair into an intractable carapace. Under such circumstances it may be necessary to begin by soaking with olive oil to which some paraffin (about one-third) has been added. If the hair be long it must be cut, and the clipping completed evenly after the crusts have been removed; it should be kept quite short throughout the whole treatment. The parasiticide most generally useful is sulphur. To the scalp, when the hair is dry, it is best applied in ointment (gr. x to ℥j, increased gradually), combined, if the skin be irritable, with zinc oxide. A powder in some cases, especially if there be much oily or serous secretion, suits better (℥ss to ℥j of fine talc powder with about gr. xv of borax), or the sulphur may be applied as a lotion (℥j to water ℥j, shaken before use), well rubbed into the scalp with a brush. In either case the precipitated sulphur forms a cake which must subsequently be removed. Salicylic acid is a useful addition both to ointments and powders, especially when secondary infections exist. As a substitute for sulphur, or in alternation with it, mercurial preparations may be used. Of these, white precipitate or yellow oxide (gr. v–x to ℥j) in ointment, with vaseline, or lanoline softened by the addition of olive oil, are, perhaps, the most convenient; or perchloride solution (1 in 2,000) may be rubbed into the scalp. Naph-

thol ointment, rendered almost fluid by the addition of oil, is also a good preparation.

Lichen is a term very loosely applied to papular eruptions, especially in children. It is usually a cloak for ignorance and Malcolm Morris[1] proposes to limit its use to lichen ruber planus, a disease resembling psoriasis, but due probably to a peculiar inflammation of the skin starting from the sweat glands. It is of such rare occurrence in childhood that it need not be described here. Lichen strophulosis is a form of miliaria not uncommon in infants. Lichen simplex and lichen agrius are stages of eczema, and lichen urticatus a form of urticaria (*q. v.*) common in children.

Miliaria or sudamina are produced by obstruction of the sweat ducts; the sweat unable to escape is effused beneath the horny layer, producing a small vesicle. Sudamina are most apt to appear after the sweat function has been arrested for a time, as by fever. They are usually most numerous on the front of the chest and the abdomen. They disappear in a few days, leaving no trace. Should inflammatory reaction take place about the sweat gland, owing to the retention of the secretion, a bright red papule is produced. The term *miliaria rubra* is applied to this condition, which is not uncommon in infants who are clad too warmly, especially if the material next the skin is irritating. This form of miliaria was formerly called strophulus, and is commonly known as "red gum." Sometimes the papule is crowned by a vesicle or pustule, but as a rule these do not rupture, and each individual spot disappears in a few days. Attention to the clothing and the use of an antiseptic dusting powder is, as a rule, all that is required.

True **eczema**—that is to say, to quote Morris' definition, "a catarrhal inflammation of the skin originating without visible external irritation, and characterized in some stage of its evolution by serous exudation"—is a rare affection in childhood. The vast majority of cases commonly called eczema are, in infants and young children, examples either of seborrhœa or of pyococcal infection (pyosis) of the skin. The disease when it occurs in children does not produce conditions differing in any respect from those observed in adults. The same remark applies to **psoriasis**, which may commence in early childhood. Lesions of the skin due to **syphilis** are considered in the chapter on that disease.

[1] "Diseases of the Skin," London, 1894, p. 136.

APPENDIX.

Absorption and excretion are both extremely rapid in infants and young children. It is, therefore, desirable to give very active remedies, such, for instance, as the alkaloids, in small doses frequently repeated. When the dose for an infant or child is to be fixed in relation to the dose for an adult, the calculation should be made in proportion to the quantity to be taken daily. Thus, if the quantity which an adult should take be 5 grains three times a day, and if the dose for an infant of one year be assumed to be a tenth of the adult dose, then the quantity to be taken by the infant during the twenty-four hours should be $\frac{15}{10} = 1\frac{1}{2}$ grains in divided doses during the twenty-four hours.

Various scales and formulæ have been suggested for calculating the dose for age. The following scale is perhaps as good as can be devised :—

First month	$\frac{1}{15}$	of the dose	for an adult.
1 year	$\frac{1}{10}$	"	"
2 years	$\frac{1}{8}$	"	"
3 years	$\frac{1}{6}$	"	"
5 years	$\frac{1}{3}$	"	"
10 years	$\frac{2}{3}$	"	"
14 years	the same dose as for an adult.		

A more accurate method would be to base the proportions on the relative weights, but this is not practically convenient. Bolognini has suggested the following formula, which is based on the ratio of the average weight at various ages to the average adult weight :—

$$d = \frac{2 + a}{25}$$

where d = dose, and a = years of age. For infants under one year the formula is :—

$$d = \frac{1}{20 - m}$$

m = number of months.

These scales and formulæ are of use as affording general indications, but there are many exceptions. On the whole, the tendency from their use would be to give rather too high doses of alkaloids, especially opium and its derivatives, and too small doses of laxatives and antiseptics.

The following notes may be of service, but the doses mentioned may often be exceeded.

DAILY DOSES.

Alcohol (brandy).—At 1 year, ℥j to ℥ij ; at 3 years, ℥vj to ℥j (daily).
Antipyrin.—At 1 year, gr. ij to iij ; at 3 years, gr. vj to viij (daily).

Belladonna.—Large doses are well borne, and must be given if the physiological effect is desired. Of the extract, gr. $\frac{1}{8}$ for an infant; for a child of 5 years, gr. $\frac{1}{4}$ (thrice a day).

Bismuth.—Rather large doses are necessary in intestinal disorders. Of the subnitrate gr. xv for an infant aged 1 year, and about twice this quantity for a child of 5 years.

Bromides.—Potassium bromide, at 3 months, gr. jss to ij; at 6 months, gr. iij to iv; from 1 to 3 years, gr. v to x; from 3 to 5 years, gr. x to xv. Of ammonium bromides doses half as large again may be given. When bromides are really required, larger doses should be given without hesitation until the desired effect is produced, but the dose must be increased gradually, and stopped as soon as possible.

Calomel.—As a purgative in a single dose (not suitable for infants under 6 months as a rule): under 1 year, gr. $\frac{3}{4}$ to j; at 2 years, j to iij. The purgative effect of calomel differs very much in different individuals, as a rule it acts more readily in those disposed to be fat. As an antiseptic, under 1 year gr. $\frac{1}{20}$ to $\frac{1}{12}$ every two or three hours to 5 or 6 doses. Above this age the doses may be doubled and the course continued rather longer.

Chloral.—Under 6 months, gr. j; at 1 year, gr. iij; at 2 years, gr. viij; at 5 years, gr. xv (daily doses).

Opium.—Tincture: under 1 year, ♏j to ij in divided doses. It is not an hypnotic suitable for infants and young children, but is valuable in minute doses as an intestinal sedative.

Quinine.—Well borne as a rule. Of the sulphate or hydrochlorate gr. ij to iij for an infant, of the tannate gr. iv to vj.

Potassium chlorate.—At 1 year, gr. jss; at 2 years, gr. iij to vj.

Potassium iodide.—At 1 year, gr. ij to iij; at 2 years, gr. iij to vj; at 5 years, gr. x (daily).

Sodium salicylate.—At 1 year, gr. iv to v; at 3 years, gr. vj to ix; at 5 years, gr. xij to xv. To be given in divided doses every 3 or 4 hours for thirty-six to forty-eight hours, except under special circumstances, when the effect should be watched.

PRESCRIPTIONS AND RECIPES.

The following prescriptions and applications are referred to in the text, but it has been thought more convenient to bring them together here:

Simple Linctus—

(*a*) Acid:	Tr. Camph. Co.	♏ij–iv.
	Acid. Hydrochlor. Dil.	♏j.
	Vin. Ipecac.	♏iij–v.
	Glycerin.	♏x.
	Aq. Caru.	ad ℥j.
(*b*) Alkaline:	Vin. Ipecac.	♏iij–vj.
	Pot. Bicarb.	gr. iij
	Aq. Anethi	℥j

Morphine Linctus—

Liq. Morph. Hydrochlor.	♏j–iij.
Acid. Hydrochlor. Dil.	♏j.
Tinct. Aurantii Rec.	♏xx.
Glycerin.	♏x.
Aq.	ad ℥j.

For children over 8 years.

Apomorphine Linctus—

Apomorphin. Hydrochlor.	gr. $\frac{1}{64}$.
Acid. Hydrochlor. Dil.	♏j.
Syrupi Limon.	℈j.
Aq.	ad ℥j.

For children over 8 years.

Bromoform Linctus (Whooping-cough)—

Bromoform.	♏ij.
Ol. Amyg.	♏x.
Mucil. Tragacant.	♏xv.
Aq. Caru.	ad ℥j

Local Applications for the Mouth—

(*a*) Pot. Permang.	gr. ij–iv.
Aq.	℥j.
(*b*) Cupr. Sulph.	gr. xxiv.
Aq.	℥j.
(*c*) Resorcin.	gr. iv–viij.
Aq.	℥j.
(*d*) Sodii Salicyl.	gr. v.
Cocain. Hydrochlor.	gr. viij.
Aq.	℥j.

Mouth Washes—

(*a*) Thymol.	gr. vj.
Boracis	℥ss.
Spir. Rect.	℥ij.
Aq. Dest.	ad Oj.
(*b*) Thymol.	gr. iij.
Sodii Benzoat.	℥iv.
Tr. Eucalypt.	℥ij.
Aq. Dest.	ad Oj.

Boric Acid Cream.

DIPHTHERIA.

Löffler's Solution—

Menthol	10 parts by weight.
Toluol	36 parts by measure.
Creolin	2 parts by measure.
Alcohol	to 100 parts by measure.

Gaucher's Solution—

Camphor	20 parts.
Carbolic Acid (crystals)	5 "
Tartaric Acid	1 "
Castor Oil	15 "
Alcohol (90°)	10 "

Dissolve the carbolic acid in the alcohol, add the camphor, then the tartaric acid, and lastly the castor oil.

CREASOTE.

Creasoti	♏ss.
Spirit. Rect.	
Spirit. Chlorof.	āā ♏ijss.
Tinct. Card. Co.	
Extract. Glycyrrh. Liq.	āā ♏v.
Mucil. Tragacanth.	♏x.
Aq.	ad ℥j.

Dose at 1 year.

HEART FAILURE.

Elixir Camphoræ (Martindale & Westcott)—

Spirit of Camphor	℥x.
Syrup	℥v.
Distilled Water	℥j.

Contains camphor gr. iv in ℥j.

Hypodermic Injection of Caffeine—

In water ℥j, dissolve sodium salicylate gr. xvj, or sodium benzoate gr. xx, and add caffeine gr. xx. Sterilize by boiling for 15 minutes. Gr. j in ♏iij.

RHEUMATISM.

Sodii Salicylatis	℥j–℥.
Tr. Aurantii Rec.	
Glycerini	āā ℥ij.
Aq.	ad ℥j.

Dose—℥j every 2 or 3 hours.

Sodii Salicylatis	
Sodii Bicarbonatis	āā ℥jss.
Tr. Aurant. Rec.	
Glycerin.	āā ℥iij.
Aq.	ad ℥ij.

Dose—℥ij every 3 or 4 hours.

Sodii Salicyl.	℥j–℥ij.
Liq. Ammon. Acet.	
Syrupi Aurant.	āā ℥iij.
Aq.	ad ℥j.

Dose—℥j every 2 or 3 hours.

Sodii Bicarbon.	gr. xxx–xl.
Potassii Acetatis	gr. x.
Aq.	℥ss.

In effervescence with citric acid (gr. x) or fresh lemon juice (℥jss) every 4 hours, to be reduced after 24 hours.

Quinine and Alkali (Garrod).

Quininæ Sulph.	gr. ij.
Potassii Bicarb.	gr. xx.
Tr. Aurantii	♏xij.
Mucil. Acac.	ʒss.
Aq.	ad ʒij.

(A single dose.) The quinine is rubbed up with the bicarbonate, dissolved in water, and the mucilage added afterwards.

Jules Simons's Liniment—

Extr. Belladon.	1 part.
Olei Hyoscyam.	8 parts.
Olei Anthemidis	15 "

Fuller's Lotion—

Carbonate Sodium	ʒvj.
Laudanum	ʒj.
Glycerine	ʒij.
Water	ʒix.

PHOSPHORUS.

Tincturæ Phosphori Composita (B.P.C.)—

Phosphorus	gr. iij.
Chloroform	ʒv.

Warm gently in a stoppered bottle till dissolved, and add the solution to ethylic alcohol ʒxv. Shake and keep in the dark (1 in 600).

Elixir Phosphori (B.P.C.)—

Tr. Phosphori Co.	ʒj.
Glycerin	ʒiv.

To be prepared freshly: contains gr. $\frac{1}{60}$ in ʒj. Dose for an infant, ♏x–xx.

Oleum Morrhuæ Phosphoratum—

Ol. Phosphorati (B.P.)	ʒij ♏xl.
Ol. Morrhuæ	Oj.

Contains $\frac{1}{100}$ in ʒj, which is the dose.

LOCAL APPLICATIONS.

Lotio Calaminæ—

Calamin. Præparat.	gr. xl.
Zinci Ox.	gr. xx.
Glycerin.	℈j.
Aq.	ad ʒj.

Lotio Hydrargyri c. Acido Carbolico (Shadwell)—

Liq. Hydrarg. Perchlor.	♏xx.
Acidi Acetici Dil.	♏xl.
Ol. Terebinthinæ	ʒij.
Sol. Ac. Carbol. (1 in 40)	ad ʒj.

Liq. Chlori—

Potassii Chloratis	ʒiij.
Acid. Hydrochlor. (fort.)	ʒj.
Aq.	ad Oj.

Add the acid to the chlorate in a large bottle ; when the chlorine given off has displaced the air add the water gradually, corking and shaking the bottle after each addition. (Should be made as required.)

Spirit of Soft Soap—

(Spiritus Saponis Kalini.)

Half a pound of soft soap is mixed thoroughly with 4 fl. oz. of rectified spirit, strained through muslin, and scented with oil of lavender (5 drops).

Ung. Chrysarobin. Co. (Unna)—

Chrysobine	5 parts.
Salicylic Acid	2 "
Ichthyol	5 "
Simple Ointment	100 "

Dusting Powder—

Zinc Oxide, Boric Acid, Starch powder, equal parts.

Antiseptic Dusting Powder—

Zinc Oxide, Boric Acid, Starch powder, Mercury Subchloride, equal parts.

FOODS AND BEVERAGES.

Cream Mixture—(Meigs modified by Rotch).—Cream (about 15 per cent.), 2 parts ; milk, 1 part ; lime water, diluted with ¾ water, 2 parts ; solution of milk-sugar (3¾ drachms, water 3 fl. oz.) 3 parts. Or cream (20 per cent.), ʒjss ; milk, ʒj ; water, ʒv ; milk-sugar solution as above, ʒiijss.

Egg Water.—The white of an egg stirred into 4 to 6 fl. oz. of boiled water, and sweetened with white sugar or a solution of milk-sugar.

Whey.—After the milk has been curdled with rennet, the curd should be beaten up with a fork and the whey strained off through muslin. *White Wine Whey* is made by adding 2 fl. oz. of sherry to half a pint of milk just at the boiling point. The mixture is then boiled for two minutes, and afterwards allowed to cool in a basin. The whey may be poured off, or strained off as directed above.

Raw Meat Juice.—Mince fine ¼ lb. best rump-steak, free from fat and gristle, add two tablespoonfuls of water, stir, and set aside for one hour. The juice is expressed through muslin by twisting. From 2 to 3 fl. oz. may be given in twenty-four hours. It may be given in milk, the taste of which it does not much modify. (Cheadle.)

Raw Meat Pulp.—Take 2 oz. of best rump-steak, scrape fine with a knife on a cook's board, removing all gristle and fat. If not quite pulped, pound in a mortar. May be taken alone, mixed with a little finely-minced parsley (about half a teaspoonful), or spread between thin slices of bread. At 1 year this quantity may be given during the day.

Fresh Lemonade.—Rub two or three lumps of white sugar on the clean rind of a lemon, squeeze out the juice, and remove pips and shreds; place together in a jug with a bottle of soda-water or an equal quantity of boiled (cold) water.

Imperial Drink—

Cream of Tartar	℥ss.
1 Lemon cut in slices	
White sugar	½ lb.
Water	Oiij.

Mix together and let them stand for half an hour.

BATHS.

Warm Bath and Pack.—The bath should be large in proportion to the size of the child, and a large quantity of water (at first at the temperature of 95° F.) should be used. During the bath, which should last 20 minutes, the temperature of the water should be raised to 104° F. or 105° F. by the careful addition of hot water. The child should be kept immersed up to the neck, and a blanket should be thrown loosely over the bath, and held round the neck. Meanwhile, a bed is to be prepared thus:—Turn down the bed-clothes, and put a blanket on the bed, so that it projects a little over the foot. Immediately before the child is to be taken out of the bath, a fairly thick sheet, thoroughly wrung out of hot water, is placed over the blanket. The patient is now lifted out of the bath, and laid on the sheet, in which it is tightly wrapped up, with the arms inside; the part of the sheet which projects beyond the feet is folded and tucked firmly under the feet. In adjusting the sheet, care must be taken to avoid creases, and to tuck it in firmly round the neck. The blanket is now folded round the patient, great care being taken to avoid any looseness or irregularity by which air could enter. The bed-clothes are now pulled down, and tucked in firmly at the foot, sides, and neck, so as to exclude the air. Profuse perspiration commonly begins in a short time. The patient should remain in the pack about an hour. The pack is then undone, and the patient quickly rubbed down with a warm, rough towel, put into a second bed (which has been well warmed), if such be available, and covered with plenty of light, warm bed-clothing. The shift from the pack to the bed must be done as rapidly as possible. In mild weather the window may be open while the patient is in the pack, but draughts should be avoided. (Jürgensen.)

Bran Bath.—Take 2 oz. bran for each gallon of water, enclose it loosely in a muslin bag, and allow it to soak for 10 minutes; stir the water with the bag, and remove.

Starch Bath.—Potato starch, ½ oz. to each gallon of water.

Linseed Bath.—Linseed meal, ½ oz. to each gallon of water.

Alkaline Bath.—Sodium Bicarbonate, ¼ oz. to each gallon of water. About 1 drachm of borax for each gallon may be added with advantage in many cases.

Mustard Bath.—Mustard, 1 oz. to each gallon of water (at 98° to 106° F.).

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